THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

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THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

JUNE, 1912

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EXHIBITION AT CLINIC OF CASES PREVIOUSLY OPERATED UPON

With Comments, Photographs, and Skiagrams

1. This is a case of bone transplantation, and I am going to make the first dressing. (See Fig. 39.) This was a case of non-union (described in the April number, p. 135). This man came from the Canal Zone with a non-union of his tibia, in which we transplanted a large piece of tibia. The exact dimensions are about ½ by ⅝ by ⅜ inch. That is the size we usually endeavor to get. We took a piece of bone for the transplant from the crest of the other tibia. Let the record show that we have primary union on the side where the fragment was removed. Let the record further show that there is a complete primary union of the wound in the other leg; that the stitches are not disturbed. The wound is in perfect condition up to this time, and as so many days have elapsed with a normal pulse and normal temperature, it should stay in perfect condition.

This dressing is carbolated gauze, and it is only in bone and joint work that we use it. As we have so much oozing, the carbolated gauze takes up secretions which follow operation. If these secretions should decompose when ordinary sterile gauze is used and the air comes in from above or below with motion, the patient gets a saprophytic infection of the skin around the wound, and that may last for days and disturb the skin approximation. If it disturbs the skin approximation, then you can have a deeper disturbance later, and you can have a pyocyaneus infection and other varieties occurring deep in, and in all these cases you have more or less hazarded the flap, and if you have necrosis of the flap, you have exactly the same condition of superficial infection.

2. Here (see Fig. 40) is another case in the same line, un-

united fracture of humerus (described in the April number, p. 193), but a little further advanced. This young man came in with a Lane plate. He had splendid apposition, but not the slightest effort at union. We took out the bone-plate and permitted healing to take place. As soon as healing of the soft parts was perfect, we implanted a large fragment of bone from his tibia, which you see. We put it in the medulla, using the rongeur to ream it out. We took a piece from the crest of the tibia, pushed it away down in the lower fragment, and when we had it ready, we took it with forceps and drove it back up into position, so that it spans the line of fracture. He has an interosseous splint, giving great strength and force, so that in handling the arm at this time you can hardly see that he has any evidence at all of having had a broken arm. It is too early for bony union or regeneration across the line. His musculospiral nerve is not involved at all, although the trouble is in the musculospiral zone. It will take some weeks for that to ossify completely. The Haversian canals in the transplant support the vessels that go above and below from the living bone, and they coalesce across the line, and this fragment will be entirely removed or will disappear, and new bone, first, of that size and shape, will be produced, and then, second, new bone which will take on the size that is necessary for support. There is one, you see, in the tibia. The case came in a rather bad condition. There was a fragment placed across. Here it is before operation with perfect apposition, but no effort at union. Here it is again, as shown in this skiagram, with perfect apposition. Here it is with the wire turned in and the splint inserted. There [exhibiting skiagram] it is with union. We can see no trace whatever of the transplant.

3. Here is a photograph of another case of the same class with the bone-plate in position. We could not swing that around without trauma. We filled in with a graft. Here is a picture showing the graft, very close and very completely [indicating], except the line of periosteum, showing that it is a detriment to the reproduction of bone in this way; it prevents the Haversian vessels from passing from the side through into the

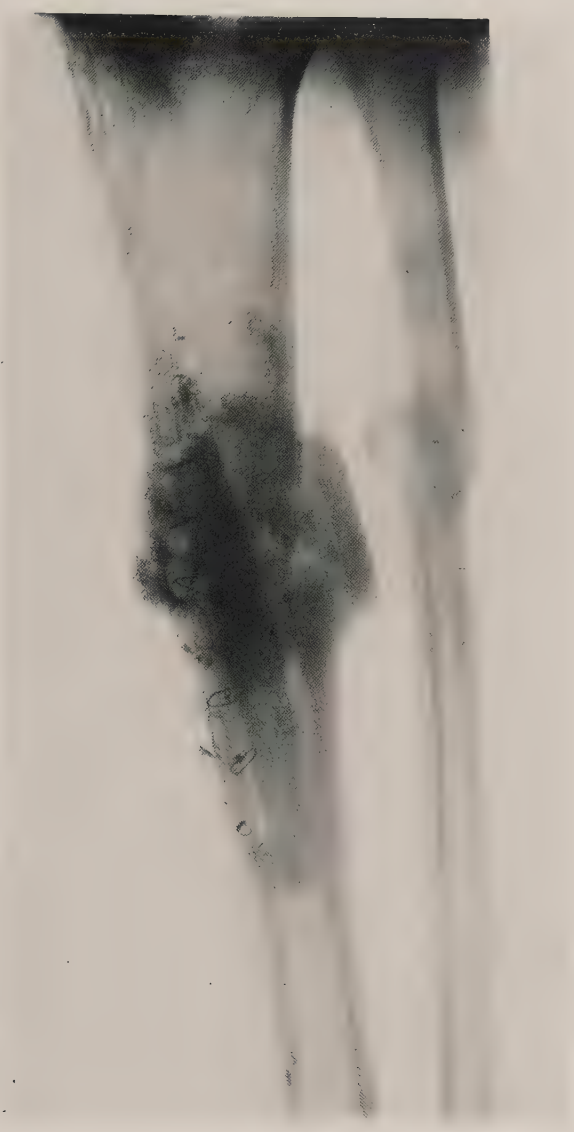


Fig. 39.—Skiagram showing case of ununited fracture of the tibia eighteen days after operation.

Fig. 40.—Ununited fracture of humerus after operation, showing bone splint transplanted from tibia.

Fig. 41.—Skiagram showing Case No. 4. Sarcoma, involving upper end of tibia. This shows transplanted bone and beginning regeneration.

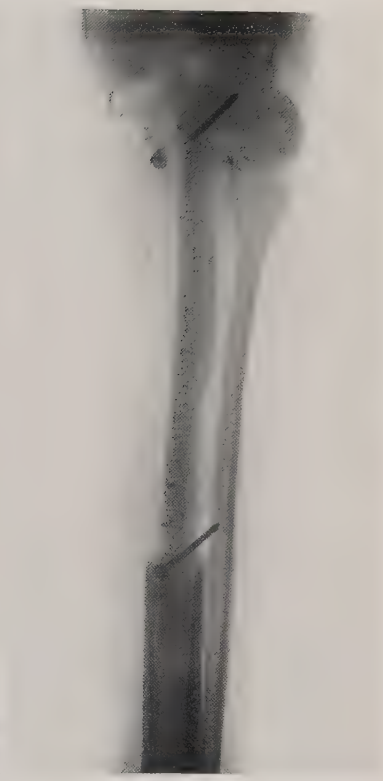


Fig. 42.—Skiagram showing Case No. 4. Sarcoma involving upper end of tibia. This shows a still greater degree of regeneration of transplanted bone.

Fig. 43.—Skiagram showing Case No. 5 in exhibition of cases. Sarcoma of the trochanter following an injury. This shows transplantation of bone after resection and its beginning regeneration.

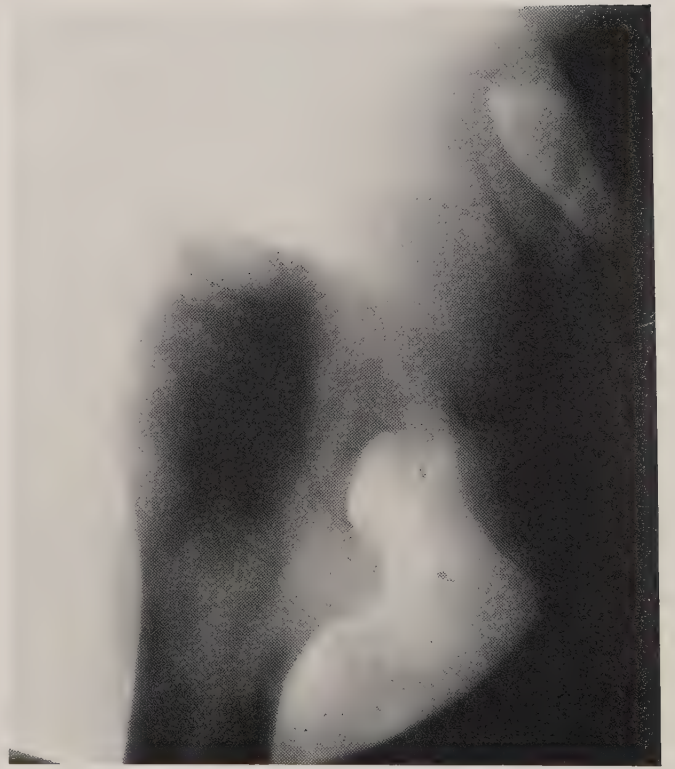


Fig. 44.—Skiagram showing Case No. 5 in exhibit of cases, etc. Sarcoma of the trochanter following an injury. This shows the sarcoma in great trochanter.

Haversian canals, and it looks as if it lived for a much longer time than we believed it would live. We do not know that it reproduces bone here. If you peel the periosteum partially and leave it attached to the shaft of the bone, it will reproduce bone. If you detach it and leave it an inch away, it will produce bone for a time, some of which will remain and some will become absorbed. There is no doubt but that living periosteum has living osteogenetic elements, that it plays a secondary rôle in bone regeneration. In bone transformation you have one requirement, so far as the regeneration of bone is concerned, namely, that the graft contacts accurately with the osteogenetic freshened bony surfaces, either one or two ends. We have here to deal with only one. The rôle it plays is that the vessels pass from the living into the Haversian canal of the dead transplant and regeneration follows. We know that if you put a fragment of bone as large as that into a muscle with the periosteum on, it will all entirely disappear with time. If you will put one end in contact with bone, it will fill up with bone of the size and shape of the graft. It will subsequently somewhat absorb if you have no particular purpose for it in the way of support.

4. This case was one of sarcoma involving the upper end of the tibia. We had just enough of the tibia left beyond the epiphyseal line to be able to cut it off and retain an articular fragment. You see this picture does not show it because it was cartilaginous, and it does not show cartilage at the line of suture. The joint was opened in doing the operation. Bony elements have been reproduced here already. It has smelted into one piece of bone, so that you cannot tell which is the transplant and which is the rest. This operation was done in lieu of an amputation for sarcoma. Formerly, for this type of sarcoma the limb was amputated. This was a slowly growing encapsulated chondrosarcoma, most malignant of this type except the giant-cell. This is the type of sarcoma you have occurring in the mandible, and occasionally in the compact tissue of the tibia and femur, and you have reproduction.

5. Here is another case, the photograph showing involvement of the joint (Figs. 43 and 44). This is a case of sarcoma

of the trochanter following an injury. We took out the upper end, head and all, and transplanted into the upper end this fragment of tibia from the patient. An autogenetic graft from the patient is by all odds the best. · You see the bone has filled clear up to this point I show you. The next picture shows it to greater advantage. When you palpate that, it feels as though you had full and perfect reproduction of bone; but a skiagram never shows young bone early. When a case of fracture of the leg is let out of the hospital, the rule is you can see through the line of fracture all the time while the patient is in the hospital or until he is discharged, but it is united just the same, although the deposit of calcareous material is not sufficient to interrupt the transmission of the *x*-ray.

6. Here is a case which gave us much worry as to whether the leg would live after we had done an arthroplasty on the knee. This woman had an ankylosis of the knee for thirty years, and the heel was up in this position close to the buttocks [indicating], and this photograph shows the bowing that occurred in the tibia. The ankylosis occurred when she was a girl of seven. Notice the curve that has formed. This is a lateral view at the point of ankylosis. We brought that around, and in doing so we put enormous tension on the popliteal structures. We exposed the arteries and veins and nerves, but all three are now in position. The next few days following the operation she had edema and swelling and cyanosis of the foot, so that I thought the leg would surely die, but it lived. Here is an *x*-ray picture after the operation (Figs. 45–47). In twenty-four hours motion and sensation had both disappeared on account of the stretching of the popliteal nerves, so that she has now paralysis of all the muscular groups below the knee. Her sensation has already come back. It is only a question of a short time when motion will return. These nerves are not cut. She will get complete restoration of both motion and sensation. I show you the degree of motion she has already. She is going to have splendid motion. She has a little necrosis of the skin, and this we will cover with scarlet-red ointment. She has a perfectly movable patella, and it is only a question of time when she will have a knee flexing to right angles. Remember,

Fig. 45.—Skiagram of Case No. 6 in exhibit of cases, etc., showing ankylosis of knee-joint.

Fig. 46.—Skiagram of Case No. 6 in exhibit of cases, etc., showing results after operation.

Fig. 47.—Skiagram in Case No. 6 in exhibit of cases, etc., showing results after operation.

this quadriceps muscle has been out of commission for thirty years, and all this will be developed as she commences to exercise.

7. The next case is one which involved a new principle, that is, of taking a flap from a distal portion, entirely detaching it, and putting it in between the bones. It was all cicatricial tissue around the knee. We could not get a flap from any place in the neighborhood of the knee to put in, so we took the aponeurosis, that is, the fascia lata, which covers over the trochanter, and implanted it in this case. There was complete bony consolidation, with almost complete absorption of the patella. You see the abduction of the leg. You see the graft. There is complete bony separation, showing a splendid result. You could not tell this from the normal knee except for the little exudate and wire around the side and the smaller patella (Figs. 48–52).

That settles the principle of fascia transplantation from a distal portion. We have completed the last link in arthroplasty. Of course, variations in technic will be made, but the principle is established. We do not claim much, but we do claim credit for establishing this principle. The field for arthroplasty is enormous; in fact, one is surprised at the vast field there is for this class of work. It is very much like appendicitis. In the early days, when appendicitis was brought to the notice of the profession, I remember reading a paper before the Chicago Medical Society on that subject when the dean of the medical profession of Chicago, who was president of one of the first-class medical colleges in Chicago, not according to Flexner, but still true, and I reported eight cases of appendicitis in that paper in 1889. He said, "How is it that a young man, who has only been in practice a short time, has had eight cases of appendicitis (perityphlitis) in a few months, while I have been engaged in active practice for over twenty-five years and have not seen half of that number?" It simply means he could not see or recognize them. They did go to the doctor. And it is just the same with these cases of ankylosis. The woods are full of them, and the field for doing this work is enormous, and it is for the younger men or our older men to perfect themselves in the technic, because the anatomic principle is definitely established for every large joint in the body, and the results are assured if the technic is carried out properly.

The avoidance of infection is the all-important matter in this work, and it is a work in which infection is more difficult to avoid than in the peritoneum, because the tissues have not the power of resistance that the peritoneal tissues have. When infection does take place, however, it is not so dangerous as it is in the peritoneum. We have only had one fatal case. We have had no return in any of the cases we have been able to trace up to date, that is, no return of bony union. We have one in which we believe there is a return of bony union. This was an elbow case. The patient was a woman who was operated on in the early period, and she would not stay in the hospital to have massage because it hurt her. In the fatal case, we operated on one hip of a man with double hip-joint ankylosis. After he was able to leave the hospital he went home and came back three months later for the second operation. He had the morphin habit. We thought we had cured him of this, but on the tenth day, after the second operation, he had a severe pain, pulled the dressings off one night, and the wound was wide open. He got suppuration, and died three weeks later from sepsis. It is the only death we have had in all the cases. In other cases we have had saprophytic infection, but not a pyemic infection and rarely superficial infection.

8. This is a case of double ankylosis of the hip. The patient was ankylosed from his knees up to his second cervical vertebra. When he got to the side of his bed he had to fall into it. Here is a photograph showing both hips ankylosed in the position you see. Here is another photograph after the first operation, and here is still another after the second operation. I want to show you the line of incision. You will observe that he has perfect freedom of motion. See what a perfect joint he has. Here is the other one that has just been operated on. This patient works his limbs day and night—so much so that I believe he moves his legs in his sleep. It was eight weeks ago since the last operation was done. This man will be a producer now instead of a burden to society. He will be able to walk without crutches and without a cane.

9. In this clinic we are compelled to have "befores and afters."

Fig. 48.—Photograph of Case No. 7 of exhibit of cases, etc. Condition and abduction before operation.

Fig. 49.—After operation, showing where the fascia lata was taken for flap.

Fig. 50.—Skiagram in Case No. 7 in exhibit of cases, etc. Ankylosed knee that had previously been operated on. *X*-ray before operation, showing complete ankylosis.

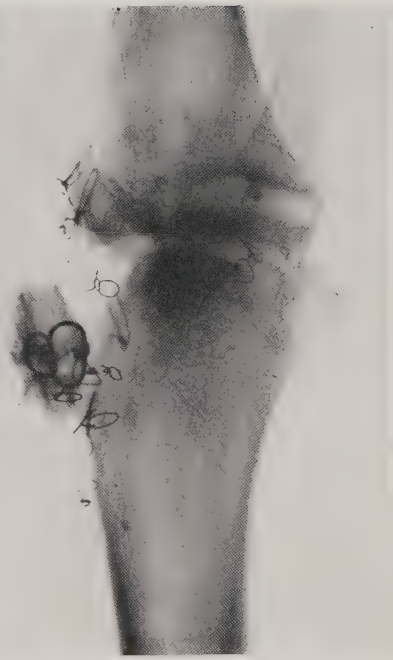


Fig. 51.—Skiagram in Case 7 after operation.

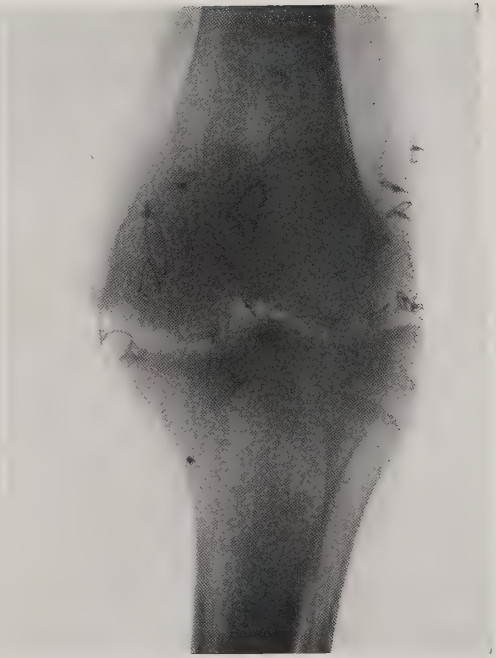
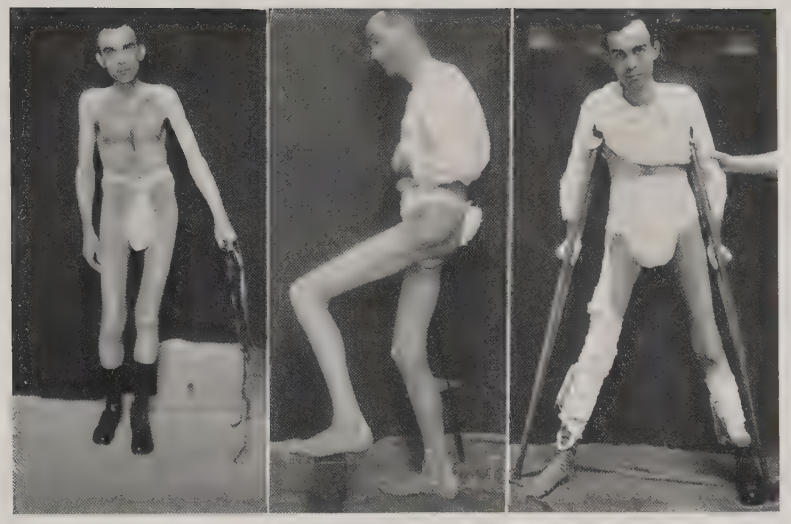


Fig. 52.—Skiagram in Case 7 after operation.



Before operation. After first operation. After second operation.

Fig. 53.—Photograph of Case No. 8 in exhibit of cases, etc.

Fig. 54.—Skiagram of Case No. 8 in exhibit of cases, etc. Showing one hip after operation.

Fig. 55.—Skiagram of Case No. 8 in exhibit of cases, etc. Showing bony ankylosis before operation. Both hips are practically the same.

This is one of the "afters." We operated on this man on the tenth day of November, 1909, for duodenal ulcer. It was one of four cases of duodenal ulcer from the same town which came in in three months. The town has a population of only 2800. The ulcer was easily demonstrable at each of the four operations. The ulcers were all diagnosed and correctly by the local doctor, much to his credit. When this man presented himself for operation he weighed 162 pounds. He now weighs 230 pounds. He said that he had been feeling bad for from fifteen to eighteen years before he was operated on. This is one phase of the results in operation for duodenal ulcer.

Unfortunately, it has another side. It is not all sunshine in these cases. The older the ulcer, the larger the ulcer, the more severe the lesion, the greater the relief and the longer the relief continues. That is a peculiar thing. It is just like the removal of the kidney. If you take out a kidney that has only a little of its tissue destroyed, you hazard the life of the patient 30 per cent. —that is, 30 in 100 die. If you take out a kidney, of which little is left,—only a shell,—you hazard the patient about 2 per cent. It is exactly the same with these cases of duodenal ulcer. If you have a severe old lesion with cicatricial contraction and do a gastro-enterostomy, as was done in this case, you get a perfect result, and these patients go around shouting your praises. If you have one with a small ulcer outside of the pylorus, or a small ulcer just inside the pylorus, and you do a gastro-enterostomy, in a considerable percentage of them you have distressing symptoms at a later period, and the question that is going to be settled is right up to the operators now, namely, that in cases in which there are extensive ulcerations and adhesions there is no indication for excision; in the first place, it cannot be done; in the second place, you do not need to do it because these cases get well after gastro-enterostomy. The *little ulcers*, those just inside or outside of the pylorus, are the ones we are going to excise. We shall then close up the opening and do a gastro-enterostomy, and then I believe we will get better results.

I had in my office day before yesterday a physician who had an ulcer of the duodenum. He was operated upon a year ago

last October. For six or eight weeks he had perfect relief. Now he has exactly the same symptoms that he formerly had. He has food distress; he turns over on his side in bed and pours the food out of his stomach, and he has all the symptoms that recur following a gastro-enterostomy. The probabilities are that he will have to undergo another operation. I did not operate upon him originally. In his case I will either have to do an entero-enterostomy or a Roux operation, or with an hour-glass pylorus do probably both at the same time. He has what he calls a typical vicious circle, and I think he is right, and still it was a no-loop operation. We are all talking about and endeavoring to avoid a vicious circle. There is something about it we do not understand, and we may as well stand up and look fate in the face, because there are some things about these ulcers that we do not know and we cannot explain. Notwithstanding that, the severe cases and the late cases give splendid results. An operation can be done with little or no risk to the patient, and when operation does not afford relief, we should go right in and do whichever one of the secondary operations is indicated after the primary operation of gastro-enterostomy. We do not do the entero-enterostomy of Roux as a primary procedure. We do four cases out of five with the oblong button, because we believe in the average case it is the safest and quickest method, but there are conditions that require us to use the suture with the clamp.

10. Here is a case of surgery we are proud of—Miss X, twenty-two years of age. It was a fierce operation, but the result has justified or warranted the effort. This patient scarcely had any courage left for us to do a palate operation, because it was declined so frequently by other operators, but being a young woman, I felt that nothing was to be lost, and everything was to be gained. She had no horizontal palatal plate. There was a perpendicular bony mass, smooth on each side of the naso-oral space, running up to the turbinated bones. The gap between it was a little wider than my thumb. She had a prosthetic mass of hard rubber that went in between the perpendicular plates in the superior maxilla clear back and pressed against the posterior wall of the pharynx, so that she could talk. It was part of a dental apparatus to give

Fig. 56.—Photograph of Case No. 10 in exhibition of cases, showing results of operations.

her an opportunity to speak. We fracture the perpendicular plates of the deformed bony palates with a reverse chisel close to their basal attachment, and bring them toward the midline, making overlapping flaps of the soft palate. We succeeded admirably and have a good hard and soft palate notwithstanding her age. The operation was done four weeks ago. In addition to that, there had been an effort made to close the lip, and it was closed at the angle of the nose, and one could put the index-finger between the two portions of the lip. We operated on her lip a week and a half ago. You can see the plan which we followed. We cut the lip in the manner I show you, swung it round, and took this portion of the nose-flap and spread it across, making a perfect lip, as you see. Look at the thickness of it. This patient has a splendid result. When her teeth are put back she will be able to speak and swallow and do everything that other people do. The lip is full length and full thickness of the normal lip and will have the same mobility.

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IMPACTED COLLES' FRACTURE

HISTORY

THIS patient is a teacher, aged twenty-one. Entered hospital for deformity following fracture of left wrist five weeks after date of accident.

History of Accident.—While patient was walking on the ice she fell forward, catching her weight on the extended left hand. When she arose it hung limp. The left wrist was enlarged, and a prominence existed just above the joint. It rapidly became swollen. She held the hand flexed at the wrist and slightly abducted. A doctor told her it was only sprained, and put on adhesive strips. She wore these two weeks. The deformity still persisted, and she went to another doctor, who put the arm and hand in a wooden splint. She wore this one week, the doctor finally giving up the case, confessing that he could not improve the deformity.

COMMENTS AND OPERATION

This case is a very instructive one, not only from the standpoint of the deformity that is present, but from the misleading character of the skiagram.

If you look at the articular surface of the lower end of the radius in this case, you will find that you are looking onto the face of the articulation. In other words, the face is not square across the lower end, but faces decidedly backward on the lower end. The normal anatomic face of the articulation of the radius is just a little forward. That should always be in the latter position after a corrected Colles' fracture.

In this girl's case there was impaction in the Colles' fracture, and the articular surface faces backward instead of forward. That accounts for the great deformity. When you look at the posterior margin of the radius, you will see there is practically no displacement at all—there is simply an impaction. With im-

paction there is a change in the direction of the articular surface, with practically no luxation.

The history accentuates what I have said in this clinic so frequently that the most common deformity following fractures in the body is that which occurs in connection with a Colles' fracture. The reason for this deformity is not because the doctor put on an improper splint, but because reduction was not effected. It is one of the most difficult of all fractures to reduce. What is the cause of the deformity from a Colles' fracture? Colles' fracture never occurs from falling on the back of the hand, but always from falling on the palm of the hand. This fractures the radius either straight across or upward and backward at varied angles. The displacement is upward or backward of the lower fragment, with rotation or luxation of the lower end; the lower fragment is tilted backward, or there is displacement of the entire lower fragment onto the dorsum of the upper fragment.

A common method of reduction is to grasp the hand, extend it, and swing it forward. It is never fully reduced by that method. The principle is wrong, and the result is a failure. The only way the fracture can be reduced properly is to increase the deformity, unlock the fragments, push the lower fragment down, and then flex it forward. Put the distal thumb on the posterior surface of the distal fragment, and put your proximal thumb of the other hand on the nail of the first thumb, and increase the deformity in the manner I show you [illustrating on an interne]. I can break that bone if I wish. I could break it easily. If the fracture is properly reduced, the fragments remain in place without any splint at all. The reduction should never be attempted without an anesthetic. It is too painful a procedure. The patient does not let you do your work properly without an anesthetic, and the result is a failure. After it is reduced, apply a plaster-of-Paris splint along the posterior surface of the arm, applying the strips parallel to the long axis of the arm, and incircling the wrist and arm three-fifths of its circumference, or put a roller bandage on it and apply any splint you wish. The arm can be carried in a sling after reduction has been properly effected. It does not make a particle of difference whose splint you use if the

Fig. 57.—Plaster cast used for the leg, showing the manner in which the cast is cut with the Gigli saw to allow for swelling and edema of the limb. The same plan is adopted in casts used for the arm and in other portions of the body.

reduction is properly brought about. It does not need a tight retention bandage. If the patient does not sleep the night after the fracture, then your reduction is incomplete. If the patient has pain that keeps him awake, go right back, do the work over again, and make a complete reduction. First increase the deformity by overextending the hand; when the deformity is increased, push the thumb down on the lower fragment, flex it forward, and that is all that is necessary for success. Then let the patient hold the hand up; if it stays perfectly straight, the reduction is all right. If it tilts, reduction is incomplete. Run a strip of plaster-of-Paris up and down the arm, and put a little roller bandage around and keep it still. Let the plaster come up the finger base and tell the patient to use the fingers right away.

This is an annoying deformity. What impairs the usefulness of the hand is that the lower sharp edge of the upper fragment is displaced forward and presses against the flexor tendons. The flexor tendon-sheaths are very sensitive, and pressure by that sharp edge, when the patient uses the hand, is very annoying. Do not keep the wrist bandaged tightly for six or eight weeks trying to get an unreduced fracture straight, because, having committed a primary crime, you commit a secondary one by causing adhesions of the tendons over the sharp edge of the lower fragment, and, as a result, the patient has limitation of motion in addition to deformity. While patients put up with deformity very well, they do not put up with limitation of motion. A useless hand is a very serious matter for a patient.

You will notice in this case that the articular surface is tipped backward. We must divide the bone in a V shape and rotate the articular surface forward so as to stop its throwing itself back in that position. It is not to be cut straight across; we will make a little V so that we can slip it around and produce the least possible defect.

When you look at this skiagram, without thinking you would say, first, that there was no fracture; second, that possibly that is a splendid result. It is a splendid result if you fail to consider the angle of the articular surface, and I am speaking now from an *x*-ray standpoint.

I want to show you this deformity. See how this fragment is thrown back! This point of bone is pressing against the tendon, giving her pain all the time. This posterior prominence will not be materially altered. The prominence in front will be altered materially. It does not seem possible that that can be the *x*-ray effect of that hand except for the one thing—a change in the articular surface. Here is a typical deformity of a Colles' fracture. I keep looking for that to see whether it is true. It is one of the most difficult situations to figure out. I never saw it before, and would not have recognized it without the assistance of the *x*-ray. When you examine that skiagram you will say that it is an excellent result, and it is, as far as the anterior or posterior replacement of the bone is concerned, except in so far as the restoration of the articular surface is concerned.

We will expose the seat of the fracture by making a longitudinal incision, about four inches long, over the radial side of the wrist, keeping posterior to the extensor longus pollicis, cutting through the skin and fascia until we see the tendons in their sheaths. These are retracted carefully so as not to traumatize them. The bone is now in view.

I am going under the periosteum. The periosteum is thickened. I am not opening the joint. The line of fracture is seen beautifully here. There is quite an impaction. Now, we must reduce the displacement. I am refracturing the bone as near as I can in the same line, and then make the V-shaped incision mentioned. When I complete the incision, I will feel sure that I will be able to rotate the axis of the lower portion around on that upper part without the slightest danger of luxation, either anterior or posterior, and that was the purpose of making the V-shaped incision in the bone. I did nothing with the ulna except to reduce the deformity; the ulna again assumes practically its normal relationship. It is a fact that there is a shortening and internal displacement of the radius that causes the ulna to become so conspicuous. The bone will rapidly fit in that position. It is in a state of incomplete ossification, but it is in a position where rapid regeneration will occur, because the bone in the neighborhood has been prepared for the advancement of the

Fig. 58.—Skiagram showing impacted Colles' fracture.

Fig. 59.—Impacted Colles' fracture. Skiagram showing impaction with change of articulating surface upward and backward.

Fig. 60.—Skiagram after operation, showing reduction of deformity and articulating surface in normal position.

Fig. 61.—Impacted Colles' fracture. Skiagram taken after operation, showing reduction of deformity.

ossifying process. The wound is closed with catgut in the deeper layers, and horsehair on the surface. A circular plaster-of-Paris cast is then applied, in the manner previously described, and as soon as it has set it will be split with the Gigli saw. We do not disturb the fracture in the least by so doing. This makes practically only a partial cast, which is sufficient for retentive or supportive purposes, and at the same time may be spread easily and not cause any pressure paralysis to occur when the arm and hand become edematous. Too tight a cast or even a constricting cotton bandage will cause a Volkmann's or an ischemic paralysis.

FRACTURE OF THE OLECRANON PROCESS

HISTORY

SURGEON, aged fifty, entered hospital February 6, 1912, because of injury to right olecranon process.

Present Trouble.—February 3, 1912, patient was walking on icy sidewalk when he slipped and fell, catching weight on right hand with arm partially flexed. He did not strike on right elbow, but felt something snap, and on getting up found that he could not extend right forearm, and has not been able to do so since that time.

Examination shows tip of olecranon of right ulna separated from ulna and retracted upward ½ inch. Patient can flex arm perfectly, but power to extend it is gone.

COMMENTS AND OPERATION

The history which you have heard tells the story. You see he did not strike the ground with the palm of his hand; if he had, he would have had a Colles' fracture. He came down and struck on his arm so as to put tension on the triceps, and the olecranon snapped off. As you see, with the limb in the extended position, there is but slight displacement. With the limb flexed, there is distinct separation of the fragments. You can easily put the end of your finger in the gap. The question arises as to what to do in this case. In the first place, there exist practically the same conditions as in the fracture of the patella, except that the olecranon has greater osteogenetic power than the patella or than any other bone of the sesamoid group. Therefore, if you secure anything like accurate contact, where there is separation of the fragments you will have bony union. If you should dress the arm in extension and permit these fragments to unite, the upper fragment will be tilted into the olecranon fossa, so that when the patient attempts to extend the arm later complete extension will not be

possible. That is one of the dangers. It is a greater danger than the danger of separation with ligamentous union. A ligamentous union in the olecranon is more detrimental to the final usefulness of the arm than a ligamentous union in the patella. The latter gives a fairly good and serviceable joint in many cases.

About two years ago we had in this clinic a patient who fractured his patella in the Civil War. The fragments separated and separated and separated until the gap measured about 5 inches. Still, he had perfect use and perfect function of the limb. He has used it ever since the Civil War as though he had never had a fracture. That accentuates the position we took some years ago, that a patient could get along perfectly well without his patella if he had disease requiring its removal, and that the quadriceps tendon could be utilized as a flap. By splitting the quadriceps tendon upward ½ or ¾ inch from its attachment to the patella in two parallel lines, dividing this section 5 or 5½ inches above, you can turn it down and suture it to the ligamentum patellæ, securing perfect restoration of function. The patient can use the joint as though he never had had any trouble with it.

We have had some beautiful illustrations of that in cases of tuberculosis of the patella where we took out the patella and used that strip of the quadriceps tendon as a support. In our last case of fracture of the patella—a stout man—we used exactly the same means of assisting wiring of the patella. We first wired the patella by the transverse loop plan, split the ligamentum patellæ longitudinally, split the piece from the quadriceps tendon, as described above, turned it down, inserting it into the slit in the patellar ligament, and locked it in there with sutures, and we have not the slightest apprehension about that man's patella. We know that either of these means will give him all the support he needs for the remainder of his life, notwithstanding that he is a very stout man. This is the last fracture of the patella in which we used that plan. In 1904—eight years ago—we operated on a patient and used the quadriceps tendon after the plan I have mentioned, after removing the patella completely. He had tuberculosis of the patella. He has almost full degree of flexion and easily supports his weight on the leg.

Fig. 62.—Skiagram showing fracture of tip of the olecranon.

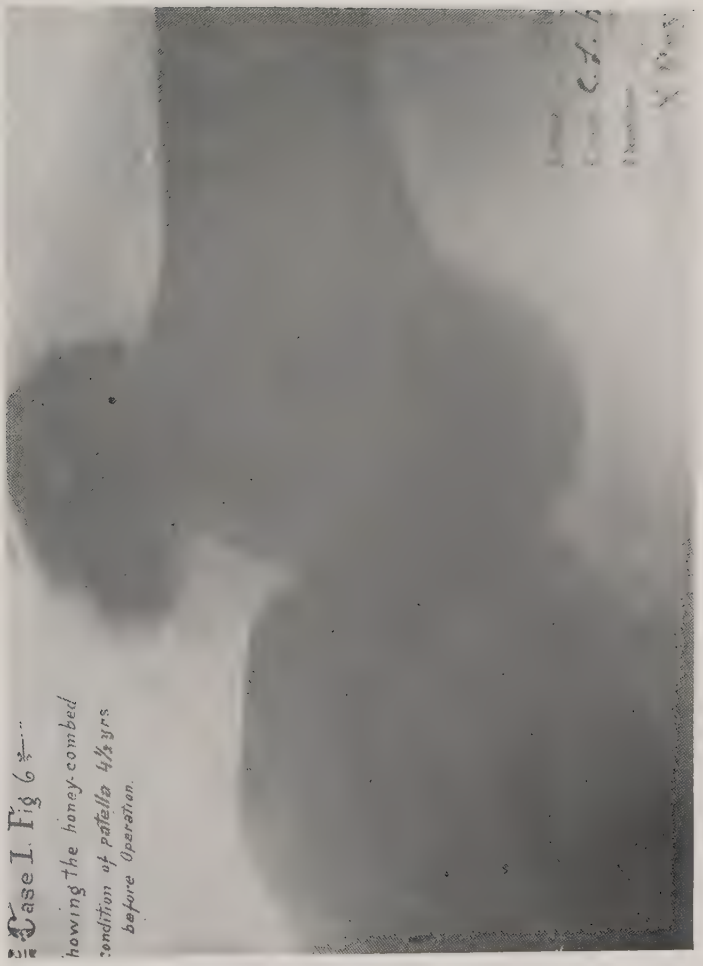

Fig. 63.—Shows condition of the patella, January 22, 1900.

Fig. 64.—Showing the honey-combed condition of the tuberculous patella six months before operation, January, 1904.

Fig. 65.—Showing normal patella in the same patient.

Fig. 66.—Shows the degree of voluntary flexion three years after operation.

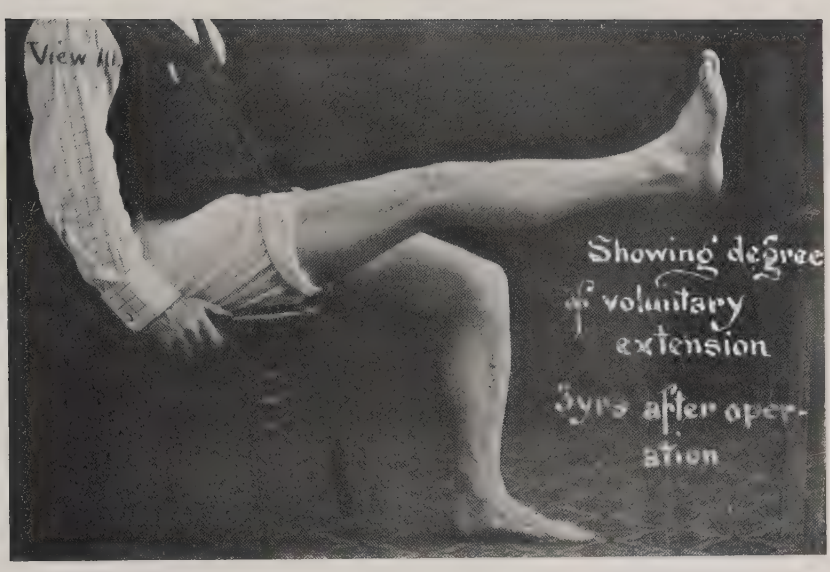

Fig. 67.—Showing the degree of voluntary extension three years after operation.

Fig. 68.—Showing patient standing on the limb three years after operation.

In all cases of fracture of the patella where we have the slightest apprehension about the subsequent result after the patella has been approximated by the circular wire loop we support it with a strip taken from the quadriceps tendon, as described. Many years ago I suggested the plan I use now, namely, of looping this patella entirely outside of the joint.

We feared the joint more then than we do now, but we never have ceased to hold a most wholesome respect for it. We never go into a joint when we can avoid doing so.

The plan I am going to use in this case is identical with the method I outlined and published in "Surgery, Gynecology, and Obstetrics" in March, 1908, and that I have used ever since, namely, wiring that button of the olecranon back against the ulna without going into the joint. This is done by making four small nicks or incisions.

The technic of the procedure is to nick the skin on the inner side and just external to the ulnar nerve. I always start from the ulnar nerve side because I can control the angle of entrance of the suture and protect the nerve all the time, and I cannot always control the angle of exit. If the drill turns from outside and you start at the ulnar side, you hazard the ulnar nerve, but, starting from the inner side in this way you never hazard the ulnar nerve. The drill is passed through the inner side through the olecranon ridge of the ulna half an inch below the line of fracture. The drill comes out clear through the skin. The skin is nicked. The drill is threaded with phosphor-bronze wire (No. 12 strand), it is drawn through, and the needle is attached to the distal side, passed under the skin above the olecranon, and on a level with the triceps tendon; it then penetrates the triceps tendon above the button of bone, comes out through a very small nick or incision of the skin, and is again passed under the skin back to the original line of entrance of the wire. The limb is extended and the wire is tied, not twisted, in the regular old-fashioned double knot—the marine knot—which gives the greatest security. We put on three knots, and then turn the edge in. That gives splendid apposition when drawn taut. The wire is buried on the inner side of the surface of the ulna, where

it is the least liable to cause irritation. If it is annoying or pricking at all, it can be removed subsequently. It should be kept in ten or twelve weeks for the mechanical support, and to avoid liability of accident, although union takes place long before that. When we originally described the technic and practised it, we found it was one of the easiest of all operations to perform in connection with the ulna, and that it involved no risk to the joint whatever.

Note.—Primary union; good functional result.

In doing the operation in this way there is no danger of exposing the joint to infection at any time, and you have a secure approximation.

In fractures of the olecranon you do not have interposition of the aponeurosis as in fractures of the patella. You cannot do subcutaneous wiring of the patella on that plan with assurance as to results, because when the patella is fractured not infrequently the capsule of the patella is torn above or below the line of bone division, and when it pulls apart, the capsule of the patella folds in between the ends. That is why an operation on the patella has to be an open operation, and why, in the case of the olecranon, we can do a subcutaneous operation.

We use phosphor-bronze wire in all these cases because it is made up of many fine strands. The size of wire we use is what is known as No. 12 strand cable, made up of 12 very fine strands. These strands are so fine that if the wire protrudes through the skin and you cut it off, the skin below that level will heal over, which is not true of any single wire that has a much larger diameter, whether it is made of gold, silver, iron, or phosphor-bronze. It is a fact that the connective-tissue cells are able to encapsulate and care for a finer wire, but they are unable to encapsulate and care for the larger strands. The only cases where we make an exception to that rule is in surgical work in the mouth. In the mouth you can put in a large-sized silver wire, because there we have the greatest type of tissue resistance and of regeneration to be found in any part of the body. We close the wound with deep catgut and superficial horsehair sutures. No drain is used. The wound is dusted with bismuth subiodid powder, and sealed

with collodion. The arm is dressed slightly flexed, a plaster-of-Paris cast being applied, but not complete. It is cut with a Gigli saw as soon as it has hardened sufficiently to allow for any swelling or edema that may occur. Passive motion will be begun in about ten days or two weeks. The cast will be removed finally in about two to three weeks. Having been cut, it can be removed and replaced as often as desired. Primary union. Good result.

DIVISION OF THE BRACHIAL PLEXUS

HISTORY

THIS patient is a laborer, forty-two years old. He entered Mercy Hospital February 5, 1912, because of paralysis of and pain in the left arm, forearm, and hand. There have been no nervous disorders among his relatives. He never has been sick in bed in his life. Five years ago he was struck on the head by a falling monkey-wrench. A doctor told him a slight skull fracture had occurred. He had no headache, was not rendered unconscious, and in two days was feeling as well as ever.

Present Trouble.—October 4, 1911, four months ago, patient, while in a standing position, was struck on the left shoulder and side of neck by a falling timber of great weight. He was rendered unconscious and remained so for ten minutes. A local doctor attended him and found the left clavicle fractured about its middle, but not compounded or displaced badly.

Ever since the accident the entire left arm, forearm, and hand have been paralyzed and are the seat of an excruciating pain. This is growing worse continually. The entire extremity has been anesthetic since the accident, save for an area on the back of the arm which is hyperesthetic. The hand and wrist become cold readily, and if allowed to hang at his side, the hand becomes congested and blue in color. The skin over the trapezius and sternomastoid muscle was anesthetic at first, but sensation has returned gradually. The right pupil has been dilated. The left pupil has been contracted to about normal size ever since the accident. Both react to light and accommodation.

Physical examination by Dr. Murphy. A pressure sensitive zone is present just where the cervical nerves of the left side come out of the intervertebral foramina. It will be necessary to expose the nerve-ends and bring them into apposition with the divided trunks.

All muscles of the arm and forearm are paralyzed, also the pectoralis major and minor muscles, the latissimus dorsi, the subscapularis, infraspinatus, and deltoid. The trapezius and supraspinatus are not involved, neither is the diaphragm. The rhomboidei are paralyzed. A hyperesthetic area exists on back of arm nearly down to the olecranon. It extends outward to the outer margin of the triceps muscle, and within and anterior to the region over the coracobrachialis muscle.

COMMENTS AND OPERATION

The history of a blow on the head is sometimes much more important than the mere fact that a patient was unconscious, or that he had a fracture of the skull. When a patient receives a blow on the head and becomes unconscious, it is a serious condition. The question arises, then, was the patient rendered unconscious? This is much more important than the mere fact that he sustained a fracture. How long was he unconscious after receiving the blow?

If you study the history of traumatic epilepsy, you will find that there is scarcely a single exception to two conditions. One is that a patient who receives a blow on the head, whether it produces a fracture or not, who is not rendered unconscious, does not get epileptic convulsions; while a patient who receives a severe blow on the head and becomes unconscious, whether there is evidence of fracture or not, later in life very frequently gets epilepsy, and the severity of the concussion and injury to the brain is estimated by the primary period of unconsciousness. When a man receives a blow on the head and is dazed for a second or so, and then is ready to go on, it really means unconsciousness. The epileptic phase is the most important phase of every injury to the head. The danger of death from a compound fracture of the skull is not so significant and important to the patient as is the danger of subsequently developing epilepsy from the injury, because these epileptic attacks come on one year, two years, ten years, twenty years, and in one case that I remember twenty-seven years, after the injury.

The question that arises in connection with this type of

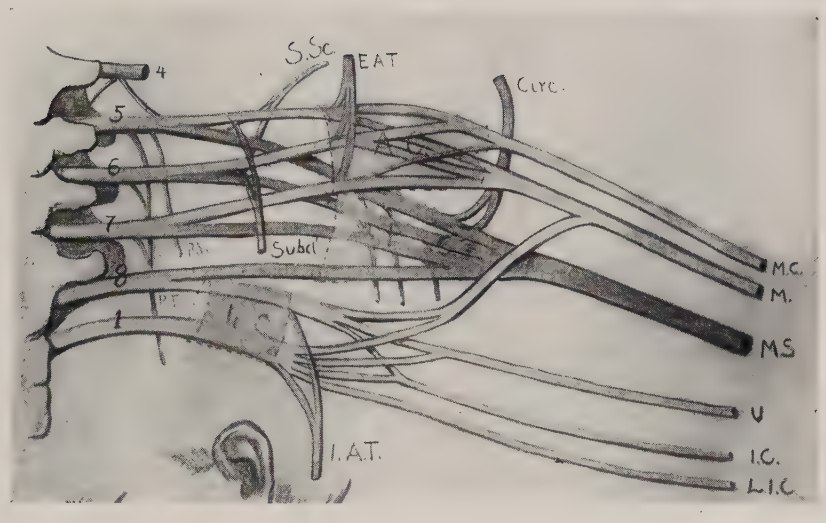

Fig. 69.—Showing brachial plexus.

brachial plexus paralysis has confronted me on four different occasions—the first time when I was a surgeon in the Cook County Hospital. A man was engaged in chopping down a tree. A second tree, which was from 6 to 8 inches in diameter, lodged against the first. When he felled the first tree the second tree fell and caught him on the side of the neck and shoulder, and produced paralysis of his upper extremity, which persisted for three years. Another case which came in later was that of a woman who was in a railroad wreck. Something in the wreck caught her on the side of the neck and caused swelling, infiltration, and edema above the clavicle. While the accident did not fracture her clavicle, she had complete paralysis of all the muscles of the arm and shoulder. The third case was that of a young man who sustained an injury also from a falling tree that caught him in the same way as the first case, with the same result. This patient is the fourth case.

The question arises in this case, Where was the real injury? Has he a division of his brachial trunks as a result of the timber crushing or dividing them, or has he a fracture of his clavicle, with a division of his brachial cords by the fractured bone? Or have the brachial roots been pulled out of the spinal foramina by traction? To reverse that order, then, we will say that if the roots have been pulled out we are not going to be able to accomplish anything. If the injury is a division of the foramina and the trunk ends can be exposed, we hope to be able to get end-to-end union of the nerve, and if that can be accomplished, after removing the neuroma we should get restoration of function. Again, is it the result of fracture of the clavicle, and did the clavicle, when it fractured, compress and cut off the brachial cords by pressure against the first rib? If the latter occurred, then it should have cut off his brachial artery, which it did not do. A pressure that would cut the nerves would surely cut the artery. The nerves, however, come in a little distance outside the artery in that position, and meet in the subclavicular position, though they meet in the upper portion of the axilla.

Again, has he had traction put on his nerves without cutting them, and has he a multiple neuromatous thickening of the trunks

—a much more serious condition than a division? A nerve that has been stretched so that its axis-cylinders are fractured in many places has developed in it a type of connective tissue through which the axis-cylinders do not subsequently pass, and which leaves a permanent paralysis. It usually extends over so long an area that excision with nerve approximation is difficult, if not impossible. It is, therefore, the most serious of all conditions that can occur in connection with an injury to the nerve. It is more common to have that type result from an injury which jerks the arm out and produces extension of the brachial plexus with luxation of the shoulder, than it is with the type that I have just described.

The operation in this case will be a long and tedious process. the question is, What nerves are involved? His phrenic on the left side is not involved. The phrenic nerve is given off principally from the fourth cervical. The fifth cervical is the first trunk of the brachial plexus. He has, in addition, paralysis of his rhomboidei. These are also supplied by the fifth cervical. He has no paralysis of his spinal accessory nerve, which receives its accessory portion from the third and fourth cervical. His trapezius is intact; it is also supplied by the spinal accessory nerve. His deltoid is paralyzed, and the only muscle left of this group which is not paralyzed is the supraspinatus, which is supplied from his fourth cervical.

We will now make an incision extending from the middle of the belly of the left sternocleidomastoid muscle downward and outward, external to the middle of the clavicle, and 3 inches below it. The clavicle is now exposed, and will be sawed through, the ends of the fragments being raised so as to expose the brachial plexus throughout its entire extent. There is a dense mass of adhesions which will necessitate careful dissection of the nerve-trunks. The phrenic nerve is not embedded in the mass. You see it responds to faradic stimulus. After we have separated these nerve-ends from the surrounding cicatricial mass, we will make an anastomosis in the usual manner by splitting the proximal end of the nerve longitudinally and using one of the pieces to bridge over the gap. We will unite the distal portion

of the musculospiral nerve to the root of the seventh cervical nerve, and the distal portion of the median nerve to the sixth cervical, using catgut for all sutures, as the retraction is so great that the distal portion of his fifth cervical cannot be approximated to its proximal end at the spinal foramen.

The trunks of the brachial plexus, made up of the fifth cervical and sixth cervical, were cut off just outside the foramen. The trunks of the seventh and eighth cervical were drawn from the roots out of the spinal foramina. The first dorsal was divided just outside of the foramen, leaving the stump on a level with the bone. The sixth cervical was freshened and united end-to-end with the distal portion of the trunk, made up from the seventh and eighth cervical, using very fine catgut sutures. The trunk of the lower portion of the median nerve was split 1½ inches below the point of division in the accident, and we turned that upward to contact with the stump of the sixth cervical. This union was supported mechanically by the scalenus medius muscle. It was impossible to make any contacting of the stumps of the seventh and eighth cervical, and they were taken out, as they had been drawn out from inside the foramen. We have, therefore, in this case the hope of securing permanent axonal contacting between the reflected area of the median and the fifth and sixth cervicals, thus continuing the nerve-current through the median to the outside filaments. This is, to all intents and purposes, a partial side-to-end approximation, which does not, as a rule, give the best results, but it was all that could be done in this particular case. We expect to have a regeneration of the axons of the middle trunk of the brachial plexus. In that way we should get some axonal regeneration in the musculospiral nerve and some in the ulnar nerve, if the suture holds, and there is not too much deposit of connective tissue. The fasciculi of nerve axons were clearly discernible in the distal portion, but were somewhat obscured in the proximal portion, even when it was divided down to the level of the spinal foramen, so that it is questionable how much axonal contacting we will get from the proximal stump of that nerve. It

will require from eleven to sixteen months to determine what the final result in this particular case will be.

The clavicular fragments are now united with catgut, and the wound closed with catgut, the skin edges being approximated with horsehair. We will put in a small drain, dust the wound with bismuth subiodid, a desiccating powder, and then apply the dressing.

The result was a primary union. He was examined on May 7th. He has practically no pain, but there is no effort at return of motion, although be believes that the area of sensation has increased.

In treating all these nerve injuries you have to consider these basic principles: First, no matter how long a nerve has been divided, if you recontact the ends without the intervention of connective tissue, you will have a regeneration in the distal axonal portions. We had one case where the nerve was reunited twenty-six years after its division and function was restored. We think that is a good time limit. Second, there must be the absolute contacting of the axons without the intervention of connective tissue, because the axons will not penetrate any considerable depth of connective tissue and establish contact except in the case of the trifacial nerve. In the trifacial nerve there is no distance that the axons will not span. You can cut out 1 or 1½ inches of the infra-orbital nerve, and it will regenerate completely. There is a fairly well-recognized law that nerves have potency of regeneration in proportion to the size of their ganglia. That is why when there are large ganglia on the large spinal roots, as in the brachial plexus, there is great potency of regeneration, and the regenerative potency of the sacral plexus is about in the same proportion. But in the trifacial you have the largest ganglion of any sensory nerve-root in the body, and in that you have a colossal potency of regeneration, not equaled in any other nerves or in any other structure in the body. The tendo Achillis comes nearest to spanning as great a distance as the trifacial nerve and have it fully restored in function. Third, after the division of a nerve-trunk there is always the death of the distal portion of the

axis-cylinder, because the axis-cylinder is an elongation of the cell-body of the neuron, and when you separate any portion of the cell-body from its nucleus, the distal portion always dies. In surgery we are acting altogether on the neuron principle, namely, the central cell-body with its nucleus, its axon, its dendrons, and its telodendrites. The dendrons, telodendrites, and axons are elongations of the cell-body itself. The axon is the central conducting wire of the neuron. The myelin sheath is not a portion of the cell-body: it is merely a cement substance, so to speak, for insulating axis-cylinders or axons. The neurilemma is not an elongation of the neuron cell-body; the neurilemma is made up of numerous elongated and elliptic cells contacting and covering the myelin sheath or the axis-cylinder. When you divide an axon, the central conducting wire dies to the periphery, fragments, and is absorbed. The myelin sheath also fragments and is absorbed. The neurilemma, which is the regenerative sheath of the nerve, becomes congested, granular, enlarges by nuclear division, and commences to reproduce a new myelin substance and to produce a new embryonal axon without conducting power. This is accomplished in from eighteen to twenty-three days after division of the nerve. The neurilemma prepares at once the connective-tissue tube through which the axon from the proximal side can pass to the periphery at any time when there is a contacting with the peripheral portion of the axis-cylinder.

It was the belief of Kennedy that there was a new axon regenerated by the neurilemma of the nerve-trunk after the division. I think Kennedy is in error in his theory. The axis-cylinder is not regenerated, but the inclosing connective-tissue tube is regenerated, into which the axis-cylinder passes from the proximal side, and regardless of whether or not Kennedy is theoretically correct, the practical fact is this, that the axis-cylinder or axon never regenerates even to the conducting of faradic electricity until the peripheral or distal segment has been contacted with the proximal living axon. So you see it does not matter whether his theory is right or not, the axon ends must be contacted, and if contacted, the axis-cylinder develops down

through the tube which the neurilemma has prepared for it, and develops again to the extreme end, supplying complete motion and sensation over the zone with which it has been contacted.

In estimating the probable result of a nerve suture, then, one has to take into consideration, first, that the ends of the axons are contacted; second, that there will be no intervention of connective tissue between these ends after they have been approximated with suture, and for that reason we embed the line of union either in muscle, which is a desirable medium, as was shown by Levings, of Milwaukee, many years ago, or we wrap it in egg-membrane. If you can, embed the reunited nerve in a muscle, which is most desirable, and sew the muscle all around it. Otherwise, use egg-membrane "as a plumber wraps a joint." This is an absorbable membrane. After you have sutured the nerve-ends together wrap the egg-membrane around the nerve across the line of union. That prevents the intervention of connective tissue from the outside. It is desirable to have a nerve-union produced by the neuroglia type of connective tissue, which permits of the transmission of axis-cylinders better than the ordinary connective-tissue type of union. The nerve-bundles are held together by neuroglia. If you get the ends approximated accurately, the neuroglia forms a definitive union of the nerve, not the connective tissue from the outside, and that is the purpose of wrapping it in muscle or egg-membrane.

There is probably no field of work in which we are at present engaged that is quite so satisfactory as regeneration of the ends of nerves when they are properly united. If you look over the literature of nerve union, you will find that, up to the time we wrote our original article on neurologic surgery, it was divided into two great schools, one claiming that a nerve, if united, will not have its function restored; the other claiming that if the nerve is united, function is restored. Both schools are right, but neither recognized the real key to the success in one or the failure in the other. The reason the men who got bad results failed was because they took the bulb-ends of the divided nerves and united them without cutting off that capsule of connective tissue which insulates the end exactly as a telephone or telegraph

cable is insulated. The men who cut off these ends and sewed the brush-like ends of the fasciculi together so as to get contact got good results. Neither, however, knew the reason for their success or failure. The result was that we had the profession divided into two great schools, one saying that nerves would regenerate when united, and the other saying they would not. Both were right, and both were wrong. The nerves would regenerate if the axis-cylinders were approximated and the connective tissue was kept out during the process of repair.

TUBERCULOSIS OF THE INTESTINES—LAPAROTOMY

HISTORY

THIS patient is a housewife, thirty-eight years old. She entered Mercy Hospital February 20, 1912, because of pains in lower abdomen. There has been no tuberculosis among her relatives. Her mother died of carcinoma of the uterus at the age of sixty-three. Her past life has been free from serious illnesses. Her menses always were perfectly normal until onset of present abdominal trouble, twenty years ago. Since then she has had some dysmenorrhea. Menses always have been rather scanty. A slight leukorrhea began at about the age of eighteen and has persisted ever since.

At about the age of twenty the patient first noticed that abdomen was larger than usual. Enlargement continued and increased steadily during the ensuing five years. Beginning at the same time as did this abdominal enlargement, at her menstrual periods only would she have severe pain in lower abdomen on the left side. It would keep her awake at night and keep her in bed always during menstrual periods. She never has had nausea or emesis, chills or fever, that she knows about. Her general health never was impaired. She had no dragging pains in pelvis and thighs. The abdomen was the size of one six months pregnant.

Three years ago she was operated on. A large ovarian cyst was removed from the left side of the pelvis. The symptoms were relieved by the operation, and her menses continued normal and without the dysmenorrhea.

Six months ago the patient first noticed that at the times of menses the pains in left lower abdomen and pelvis were beginning again. They were never present between menses, and at men-

strual periods were exactly of the same nature as those which occurred just previous to her operation three years ago.

Since July, 1911, she has noticed severe pain down in the abdomen, in the median line, at the site of the old scar. This pain occurs irrespective of her menses, and she thinks is of a different nature than the pain on the left side at her menstrual periods. The menstrual pain is much more severe. The menses have not changed in character, amount, or frequency, nor has she ever missed a period.

Examination by Dr. Murphy (Vaginal).—A double mass is present—one behind the uterus, which seems encapsulated and movable. It can be lifted up and out of the pelvis. The other is a part of the fundus of the uterus extending up toward and becoming a part of the left rectus muscle. This mass is not at all connected with the other tumor. The mass is hard, runs out to the rectus, and gives some evidence of infiltration into the neighboring structures.

From the physical signs it appears that she should be operated on to remove the deep neoplasm. One cannot say from palpation where the tumor originates. The incision must be on the right side, so as to be to the right of the mass in the rectus muscle.

COMMENTS AND OPERATION

Aside from the statement concerning this fibroid, she had a cyst—an ordinary pedicled cyst—on the left side. What date was the operation?

THE INTERNE: May, 1908.

DR. MURPHY: Now she comes back with a tumor in the abdominal wall. In addition to that she has a small round tumor in the abdomen which I am able to luxate and move upward in the pelvis, but which does not appear to be a part of the round tumor above. The uterus comes up and rests against the under surface of the abdominal wall, and appears to be part of the mass attached to the abdominal wall, although the history of the case does not indicate that there was any fixation of the uterus to the abdominal wall in the previous operation. You can see that it is an extraordinary condition, and when you come

to ask yourself, What are the things that this may possibly be? you think, first, that it might be a sarcoma; second, that it is possibly a carcinoma, where there was an adenomatous condition of the ovary, with rupture of an adenomatous cyst of the papillomatous type and infiltration of the wall. Third, it may be a neoplasm of the desmoid type, originating in the muscle of the abdomen. The desmoid tumors, however, are very rare. In all my experience I have seen only one true desmoid that was hard and woody. It feels, except on one side, like a sarcoma. At the first operation the abdomen was closed with catgut. This might be or could be a tuberculoma occurring in the scar of the former tumor, but it feels more like a sarcoma than like anything else. The desmoids are not quite so hard as the sarcomata on palpation.

I am going to open the abdomen through the middle of the rectus muscle. There is no thickening of the layers of the aponeurosis of the rectus at this point, and no infiltration of the muscle itself. We will go toward the midline in the muscle and see whether we encounter infiltration in the muscle as we enlarge our incision. There is a sense of resistance, but it appears to be on the under surface of the muscle, or at the peritoneal margin, rather than in the muscle. It does not appear to infiltrate the muscle. This looks like the properitoneal fat. Notice how cautiously I am going through. There are no adhesions at this particular point. That is fortunate. It is better than I expected. I must inspect every point within before I proceed, because I do not know just where adhesions exist. I am able to palpate and go down on the right side of this mass, and shall endeavor to make an examination of it. I will enlarge this incision upward, so that I may get around the upper margin and palpate it on the right side of the abdomen. Here is the sigmoid above the lesion. The sigmoid runs into the lesion. It is a tubercular ulcer of the intestine, of the sigmoid,—a granuloma,—and adherent to the anterior abdominal wall as far as we are able to make out. That it is a lesion of the sigmoid is easily demonstrable. There are ulceration and thickening of the sigmoid. There is no thickening or ulceration above, and there is no

mechanical obstruction. There is some infiltration. In some places on the surface you see normal areas of bowel. Contrast the two.

Should we resect that bowel? No. Formerly that was the plan. Now we do not do it because, when you commence to resect an adherent and fixed tuberculous intestine, unless you can get out all of that tuberculous intestine with a zone entirely free from adhesions in which to make a subsequent anastomosis, it would open again at once, or there would be a fecal leakage. Possibly it is an actinomycosis. I feel a crater in the intestinal mucosa from the other side. The mass in the bowel is probably a tuberculoma. We had one of these cases not very long ago. The patient had a cancer of the bowel with a metastasis in his femur. He came into the hospital on account of pain in his femur.

In this case there is a crater in the sigmoid burrowing forward at a point where it is easily noticeable above the point of fixation. It is my belief that we have here a tubercular ulcer; that it has already perforated the wall of the intestine, and that it is fixed to and has become a part of this granulomatous mass on the anterior abdominal wall.

I saw a case similar to this with Dr. Alexander Wiener some time in August, and I cite that case in order to emphasize the treatment of those cases. His patient had a very large mass over her cecum, pressing against the anterior abdominal wall. An exploration was made, I believe, but I am not positive about that. The mass was found adherent, or possibly it was not. However, that is a matter of no consequence. The result was that this mass filled all the right iliac fossa. The tuberculin test showed that she had a temperature of 103° F. She received 5 to 6 minims of dilution No. 2 of Lucius and Brüning's tuberculin. As to this mass, while it looked very much like a carcinoma, the tuberculin test settled positively the fact that it was not a carcinoma, but a tuberculoma. She has been on tuberculin treatment since August, and I learned from Dr. Wiener the day before yesterday that the tumor which I saw, and which was as large as my two fists, had entirely disappeared. It could

no longer be felt in the patient's right side. That is typical of the course in tuberculosis of the intestine under tuberculin treatment.

When you look over the history of cases of tuberculous ulcers of the intestine you will find that it has been divided into two distinct epochs, from a curative standpoint, as well as from a clinical point of view—the pretuberculin period and the post-tuberculin period. In tuberculosis of the intestine and tuberculous peritonitis, when the lesion is circumscribed, the primary indication is to resect the tuberculous intestine. But you must have a peritoneum that is free from tuberculosis when you make that attempt. You must have an intestine that is practically free from adhesions to the anterior or posterior abdominal wall. Why? If the peritoneum is infiltrated with tubercles at the time you do the operation you will, as a result of the operation, frequently have a fistula—a fistula that you will be unable to close.

I recall my first painful experience when I thought I could do better than the masters of their time—Fenger and Senn. A patient came to me at the Alexian Brothers' Hospital with a tuberculous fistula of the intestine. The patient had been operated on half a dozen times. Dr. Fenger had operated on him twice, and, I believe, Dr. Senn had also operated on him twice. I thought I could beat these men on this proposition. I had had a good deal of intestinal experience, and I thought I could follow a line I had laid down and succeed in getting union. I put it together, and put it together, and every time I put it together there was exactly the same result—failure of union and fistulous development. That patient underwent two operative procedures at my hands and went away with a fecal fistula. I learned the lesson that wherever you have the peritoneum involved in tuberculosis, you should be extremely cautious about making any opening in the intestinal tract, because it is easy to open it, but the great difficulty is to get it closed, and that is what you encounter in this class of cases. So here we will make no effort at all at removal. We will first make the tuberculin test, and as soon as she is over the effect of the operation we will continue the use of Koch's old tuberculin as a line of treatment.

You will remember, I just said that where the intestine is free from adhesions we can resect the intestine and establish end-to-end or side-to-side anastomosis, or a button union if the peritoneum is not infected or invaded. If the peritoneum is invaded by the tuberculous process, or has that slimy, gelatinous, cobwebby adhesive surface, do not touch it, because the endothelial cells have been disturbed by the tuberculosis and the intestine will not agglutinate and unite as the intestine should. If you cannot do an excision, what is the next proposition? When you have intestinal obstruction, the next proposition is to side-track the fecal current from the area of obstruction, if it be large. In other words, exclude the portion of bowel that is involved in the tuberculosis. In this case the bowel is involved so low down that, to make exclusion of it, you must establish an artificial anus, and an artificial anus is not justified except where a patient is suffering from the acute symptoms of tuberculous intoxication with a mixed infection; that is, having fever and sweats and all of that. This patient has none of that train of symptoms—none at all, and, therefore, an artificial anus is not justifiable.

The next procedure, when you cannot exclude the intestine, is to put the patient in the best possible condition you can, with the intestine remaining, and without exclusion—which is what we are going to do here, and later, unless the patient commences to improve, if she shows evidences of septic intoxication in connection with the tuberculous infection, then we will make an artificial anus on the left side, above the position where the sigmoid is adherent to the anterior abdominal wall. You can see that this condition is merely a coincidence. The sigmoid has become adherent to the abdominal wall where this patient had a previous laparotomy. She probably had some adhesions there which fixed the sigmoid and caused it to remain in that position, and that is where the ulcer endeavored to penetrate, the ulcerating sigmoid attaching itself to the anterior abdominal wall.

In treating these cases, where we cannot do a surgical procedure without removal of the intestine, where we do not wish

to perform bowel exclusion, the next best treatment is internal medication.

In 1891 Paul Goodman exhibited before the Pathologic Society in Berlin six specimens of intestines that had been affected with tuberculosis,—that is, tuberculous ulcers,—in which the ulcers had healed. This occurred in patients who had been treated with Koch's old tuberculin. You will remember Koch's old tuberculin came out the year preceding, and had been used in many hospitals, and used as it was at that time, in very large doses, they had pronounced reactions. Goodman's patient died from tuberculosis of the lung. Virchow, who was the greatest cellular pathologist of that time, said he had never, up to that time, seen a specimen of tuberculous ulcer of the intestine that had fully healed. This threw a new light on the use of tuberculin in tuberculous intestinal lesions. While he had seen lupus heal, he had never seen a tuberculous ulcer of the intestine heal. Then they started in to use tuberculin in cases of ulcer of the intestine. Tuberculin was lauded by the medical press in the treatment of tuberculosis, and was used indiscriminately and injudiciously by the profession; consequently, the results were not so good in many instances as might be expected.

Tuberculin fell into disuse. While it is one of the greatest discoveries of the time, and one of the most potent of medicaments that we have at our command, with the exception of antitoxin and antitetanic serum and the rabies serum, it has never become a popular agent in the treatment of lesions of the intestine, from the fact that it appeared to be such a failure in connection with lesions of the lung. The failure was not due to the drug having no potency, but to the fact that it was given in improper dose. It took about twenty years to learn how to dose it properly. It took twenty years to learn what the opsonins meant, and what the opsonic index meant in the management of disease with the serum treatment. Since then we have used tuberculin judiciously and injudiciously. *It is one of the agents that has to be used with a proper and intelligent knowledge of its purpose, and when this is mastered, it is a very efficient agent.* It is particularly efficient in lesions of the intestines.

Since that time there have been a number of cases of healed intestinal tuberculosis. There are now on record an enormous number of cases of tuberculosis of the peritoneum, both in the male and female, that have been healed with the aid of tuberculin. I do not say that we can *cure* tuberculosis with tuberculin, but that is the word I would use if we can use the word *cure* in connection with medicine at all. In other words, it stimulates the reparative process so that nature is able to heal the lesion. Tuberculous lesions, as you know, in every position the body, are healed by cicatrization; in other words, by encapsulation with connective tissue or by exclusion. In the earlier reports of the use of tuberculin in connection with lupus it was pointed out that it healed by exclusion, by exfoliating, and by throwing off the tuberculous necrotic infected zone, and in that way gave good results. In the intestines it cures in the same way. There is exfoliation of the ulcer surface; then there are cicatrization *plus* cicatricial contraction of that tissue and complete repair. In the process of repair it is not uncommon to have contraction so severe as to produce stenosis of the intestine.

Tuberculosis of the intestine is just like lupus; it can be divided into three distinct classes, depending upon the degree of response on the part of the tissue to the irritation of the infecting microörganism. You will find in lupus, first, a small, circumscribed tubercle encapsulated by connective tissue. First a papule, then a pustule, then an ulcer, then a cicatricial mass, and finally repair. You find in the intestine exactly the same process. You will find in lupus that while the lesion is healing in one place it is breaking down in another. You find exactly the same thing in the intestine. You will find in lupus that occasionally there is an enormous, thickened, infiltrating mass rising half an inch above the surface. That occurs more commonly in colored people, known as the hypertrophic type of lupus. You will find in the intestine the same type of hypertrophic tuberculosis, so that in these tuberculous granulomata in the cecum and in the sigmoid you can find a tumor sometimes as large in diameter as a fist, without perforation. That is the hypertrophic type of tuberculosis. That is, there is an enormous

quantity of connective tissue thrown out to defend or prevent invasion of the tissue by the tuberculous microörganism.

Then there is another type,—the circumscribed type,—in which there is a large gangrenous area in the center of the ulcer. That is what you see in lupus. With this type of intestinal lesion you can see as it advances and repairs what happens. The tumor grows in one place, a cicatricial mass develops in another, and finally the cicatricial mass becomes so great that it causes stenosis of the intestine, and later, as the process of repair goes on, there may be intestinal obstruction. The process may be postponed for weeks or years. I had one case in which it is my recollection that eleven years of the disease elapsed before intestinal obstruction was produced. Sixty inches of the small intestine were involved in the tuberculous process. We removed the 60 inches of intestine, which was free from adhesions, and got primary union, and we followed exactly and identically the precautions we mentioned today and carry out in all our cases since that time.

This patient will be given tuberculin. I believe that this is a tuberculoma, and not a sarcoma. The tuberculin will be crowded along, increased in dosage until we get a uniform reaction of 99.4°, 99.6°, and 99.8° F. the next afternoon after she receives the injection. A reaction that elevates the temperature just one degree beyond what is normal in that patient in the afternoon is just the right reaction. It is indicative of the best opsonic index and the best resistance of the patient. The tuberculin will then be pushed and pushed; the general care of the patient will be attended to—she will be urged to lead an outdoor life and all that which goes toward making for the repair of tuberculosis in other positions of the body, and no further operation will be done on her until the time comes when we may have to make a resection of the bowel on account of intestinal obstruction.

This is a most interesting case, because it is the first one of the type that I have ever seen where the tuberculoma attempted to perforate through the anterior abdominal wall in the midline. Usually it comes over the cecum as a large mass with infiltration, or it comes over on the left side just above Poupart's ligament,

and you find an adherent tuberculous mass to the sigmoid. In this case it has become fixed to the anterior abdominal wall.

The ability of the intestine to repair itself is great, notwithstanding that it is constantly in the presence of mixed infection. In estimating the destruction of tuberculosis in every position of the body you have to take into account the mixed infection element. Tuberculosis anywhere, except in the meninges, is practically never fatal, except when there is a mixed infection. Think of it! Therefore it is the mixed infection element in all these cases, whether in the lung or the intestine or on the skin, that plays the fatal rôle. There are different degrees of resistance to tuberculous invasion in the serous tissues of the body. The acme of resistance to tuberculosis is in the peritoneum; the minimum, in the meninges, that is, so far as serous surfaces are concerned, without the use of tuberculin the minimum of complete repair is in the intestine. The mucosa of the bladder will repair itself. The mucosa of the stomach will repair itself, but the mucosa of the intestine never repairs itself, according to Virchow, except under the use of tuberculin.

We are going to have about the same contest as to the value of tuberculin that we have had with every other medical agent and with every operation that has been introduced in medicine. Naturally, it is going to be more equally divided in tuberculosis because more than 50 per cent. of the people are going to use it long before they have an intelligent basis for its administration, and it will be given indiscriminately and in improper doses, and the reason for this is that we do not read carefully and we do not act with precision—*precision!* We read an article—get a smattering of its contents. We think we have grasped the author's idea, and we go to work to carry out his idea, when every step of the procedure should be gone over and over again, and, better than all, you should see the author do the operation or administer the treatment, and then try and do it better than he does if you are confident that his results are not up to the standard in his line. Do not try to do it better than he does on the second case. That is what usually happens. It is a confession, but it is true. It is a lack of precision of doing things in a systematic, regular

way. It has its disadvantages to the people and its advantages to the profession because it is that willingness to depart from prescribed lines that has made pioneers. But we should not be pioneers until we prepare ourselves for doing pioneer work in a definite line by knowing what has been accomplished in that line up to that time.

Note.—A typical response to the tuberculin test was noted. The patient was placed on the tuberculin treatment, and a report from her physician some six weeks later stated that her condition was improving.

CYSTIC GOITER

HISTORY

THIS patient is a housewife, thirty-six years old. She entered Mercy Hospital because of an enlargement at the site of the thyroid gland, progressive weakness, and a sudden loss of voice. There has been no tuberculosis, malignancy, syphilis, goiter, or nervous disorders among the relatives. Never before has she had a serious illness. Her menstrual history is perfectly normal. She has borne three children, the last three years ago, since the onset of the present trouble, but the disease seemed to be in no way influenced by this.

Present Trouble.—In 1902 the patient first noticed an enlargement under the middle of the belly of the left sternomastoid muscle. It was not large, and she gave it little thought. During the nine years following its appearance its size increased gradually, but it evinced no symptoms. One year ago its growth seemed more rapid. At times a slight sense of pressure was felt just back of the thyroid notch, and sometimes she would find it necessary to swallow forcibly several times while eating. She has been a nervous woman all her life, more especially so during the past year. She has had headaches and insomnia often during this time. Eight weeks ago the above-named symptoms became more noticeable, and she was no longer able to do her housework. She became fatigued upon the slightest exertion, and general weakness progressed rapidly, though she did not lose in weight. For the past six weeks she has been in bed a large part of the time, mainly on account of weakness. She often has headaches and pains in the back and limbs. Throat has never been sore or inflamed.

March 8, 1912, for no apparent reason, she was seized by a hard coughing spell which lasted four hours, and at its conclusion she found that the power to speak above a whisper was gone.

She had never been markedly dyspneic before, never had coughed much, and never had been hoarse before or since that time. Pulse is 80. Temperature, normal.

Laboratory Findings.—The urinary examination proved negative. The blood examination showed 4,320,000 erythrocytes, 6800 leukocytes, and 90 per cent. of hemoglobin.

Heart Examination.—The heart-sounds were regular. No murmurs were present. The heart was not materially enlarged. A malformation of the radial vessels exists, and this interferes with the estimation of the nature of the pulse, so that another vessel, preferably the carotid, must be used for this purpose.

COMMENTS AND OPERATION

Dr. Murphy: A good history is the thing that tells the story. This history, as it was read, did not tell the story—progressive weakness and loss of voice. It should be progressive weakness and instantaneous loss of voice. What a different thing that is, is it not? Just one word spoils the purpose of the history. She was coughing, and suddenly, during her coughing spell, she lost the use of her vocal cords. That is the point I want to bring out. The increase in the size of the swelling in the neck came that night. Dr. Tivnen examined the vocal cords, and informed us that they are both paralyzed. According to the history, she has exophthalmic goiter, but she has none of the symptoms of Graves' disease, the only one being progressive weakness. She has no tremor; she has no tachycardia; she has no exophthalmos—simply a sense of increasing fatigue, and the paralysis of both vocal cords, which came on suddenly. It came on too suddenly for carcinomatous invasion. How old is this woman?

The Interne: She is thirty-six.

Dr. Murphy: Early for carcinoma, still not at all so early that carcinoma can be excluded. I have under observation at present a patient, twenty-six years of age, who had a portion of the thyroid removed. The specimen was pronounced malignant by a pathologist. You can see why this could be malignant at her age, and if malignant, one of the first things it attacks after penetrating the posterior wall of the capsule of the thyroid is the

recurrent laryngeal nerve, and then she could have vocal-cord paralysis. Carcinoma does not begin suddenly, as a rule. Carcinoma rarely ever involves both the vocal cords simultaneously. See the point I am trying to make! The clinical history does not fit the clinical course of carcinoma, because in carcinoma of the thyroid the paralysis is of the slow, progressive type, terminating in a complete paralysis. Dr. Tivnen will tell you about the condition of the vocal cords.

Dr. Richard J. Tivnen.—We are all familiar with Dr. Murphy's insistence that a clear and detailed history be obtained of every case, and the great importance he attributes to the "history" as an aid in arriving at a diagnosis and the formulation of an intelligent treatment.

In the case before us the wisdom of Dr. Murphy's position on this matter is well illustrated. An analysis of this patient's history from a laryngologic point of view presents the following facts:

1. *Previous History.*—Has not previously suffered, and does not at the present time suffer, from any of those diseases which are, more or less frequently, associated with impairment or loss of the voice, such as affections of the nose, pharynx, throat, or respiratory tract, diphtheria, syphilis, scarlatina, rheumatism, typhoid fever, pneumonia, central lesions, cerebrospinal lesions, tabes, hysteria, malignant neoplasms of contiguous structures, aneurysms, or toxic causes, such as poisoning by arsenic, lead, or atropin. She has had a slight goiter for some time past, which has caused her no inconvenience.

2. *The History of the Present Difficulty.*—One week ago last Friday the patient was suddenly attacked with a violent fit of coughing, accompanied by a feeling of strangulation, smothering, and suffocation; a swelling occurred in the neck at the time of the attack, and immediately following the attack she was unable to use her voice.

3. *The Subsequent History.*—Up to the present time she has not recovered the use of her voice; the swelling in the neck has somewhat subsided, and she has experienced no further difficulty in breathing.

Examination of the patient presents the following findings: She exhibits a moderately enlarged thyroid, the right lobe slightly larger than the left; it is uniform, not tender, firm throughout, and moves freely with the larynx; nose, mouth, and pharynx normal; examination of larynx discloses both vocal cords normal in color and presenting no inflammatory infiltration; both cords occupy the cadaveric position; during respiration they are quiescent, and on efforts at phonation they make no attempt to approximate; no evidences discernible of neoplasms within the larynx or trachea; ocular examination discloses absence of exophthalmos or the presence of von Graefe's, Dalrymple's, Stellwag's, or other signs of Graves' disease; tachycardia and tremor are likewise absent; vision, excursion of lids and globe; pupillary reflexes are normal.

The history, the symptoms, the clinical course, and the objective findings in this case lead to the conclusion that the patient's attack of coughing, dyspnea, and strangulation was a manifestation of laryngeal spasm; that the aphonia which followed was brought about by inability of the vocal cords to approximate on efforts at phonation; that this inhibition of vocal function was due to paralysis of the laryngeal nerves which supply the muscles concerned in this act, and, finally, that the entire cycle of disturbance was due to the pressure of the enlarged thyroid upon the laryngeal nerves, resulting in what is known as bilateral paralysis of both recurrent laryngeal nerves, branches of the pneumogastric.

Paralysis of the vocal cords in enlargement of the thyroid gland and similar pathologic conditions are of more frequent occurrence than is generally supposed. The fact that the voice in such affections is frequently not markedly altered is exceedingly misleading, and may cause the presence of a paralysis to remain unsuspected. It is quite possible for a patient to suffer from a moderate paralysis, or a paralysis of a certain group of laryngeal muscles, and at the same time the speaking voice exhibit little alteration in volume or timber. This is explained by the clinical observation that one vocal cord may be paralyzed, and its fellow, during the act of phonation, exhibit a compensa-

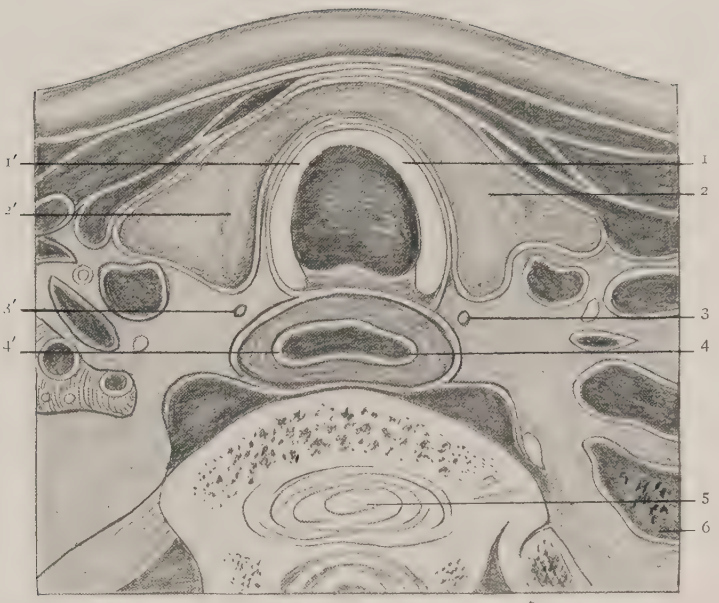

Fig. 70.—Transverse section of the neck at the level of the first dorsal: 1 and 1′, Wall of the trachea; 2 and 2′, thyroid body; 3 and 3′, right and left recurrents; 4 and 4′, esophagus; 5, vertebral body; 6, apex of the right lung.

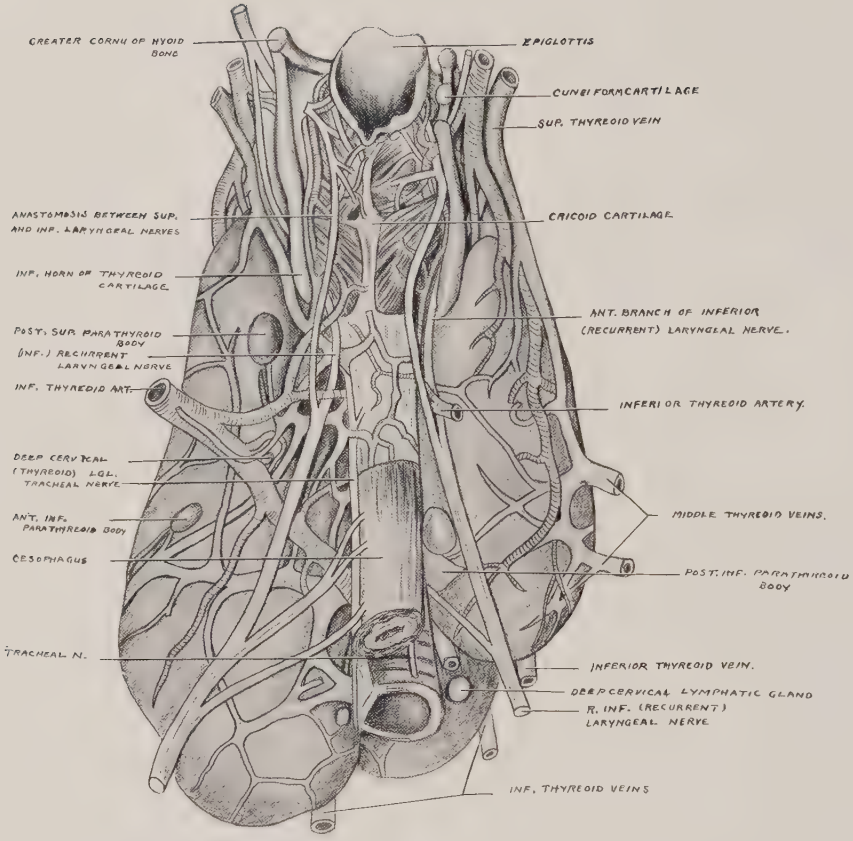

Fig. 71.—Larynx from behind—goiter. (From the body of an old woman the enlarged thyroid gland (goiter) was removed with the larynx and the trachea, and hardened in formalin; only a small portion of the esophagus remains; the posterior wall of the larynx is dissected. The large left lobe of the thyroid gland caused the trachea to rotate around its axis. The recurrent laryngeal nerve has been drawn aside on both sides, in order more clearly to show its branches) (from Bardeleben, Applied Anatomy, Rebman Co.)

tory movement sufficient to cause an approximation of the vocal lips, so that satisfactory phonation is possible.

Matthews, of Rochester, Minn., from an observation on the vocal cords in 1000 cases of goiter, found a total of 289 partial or complete paralyses. The intimate relation of the thyroid to the nerve-supply of the larynx readily explains the vulnerability of the laryngeal structures in affections of the gland.

In Fig. 73 this relation is shown very clearly. The larynx is subcutaneous throughout, while the trachea is so only at its commencement; as it nears the thorax it progressively assumes a deeper level. The thyroid gland, as we see, is situated at the upper portion of the trachea; the base of each lateral lobe extends downward upon the side of the trachea as far as the fifth or sixth tracheal rings; the apex rests upon the ala of the thyroid cartilage. Each lateral lobe averages about 2 inches in length, 1¼ inches in breadth, and ¾ inch in thickness. Occasionally a pointed lobe, the pyramidal, extends upward from the isthmus. The isthmus lies in front of the second, third, and fourth rings of the trachea. The gland is firmly attached to the larynx, and is subject to all its movements; its deep surface is concave and in contact with the trachea, larynx, pharynx, esophagus, inferior thyroid artery, and recurrent laryngeal nerve.

The capsule of the gland is well shown in Fig. 71. The recurrent laryngeal nerves in Fig. 71 are shown in their normal position, external and posterior to the capsule. These nerves are the nerves of the larynx most commonly involved in affections of the thyroid gland. As is well shown in Figs. 70 and 71, they are in intimate relation with the posterior surface of the gland, in a favorable situation to be affected by pressure, and are also a source of hazard in surgical procedures on the thyroid.

In Fig. 72 we can trace the origin and course of the recurrent laryngeals. The left recurrent is the longer, and is given off at the level of the transverse portion of the arch of the aorta, passes under it, and ascends to the larynx. It has an intimate relation to the aortic arch and subclavian; this fact and its longer course explain why it is the most often affected in pathologic processes of neighboring structures.

The right recurrent nerve is given off on the level with the subclavian, around which it curves to ascend to the larynx. Both recurrents ascend in the groove between the trachea and esophagus and end at the cricothyroid articulation by dividing into terminal branches.

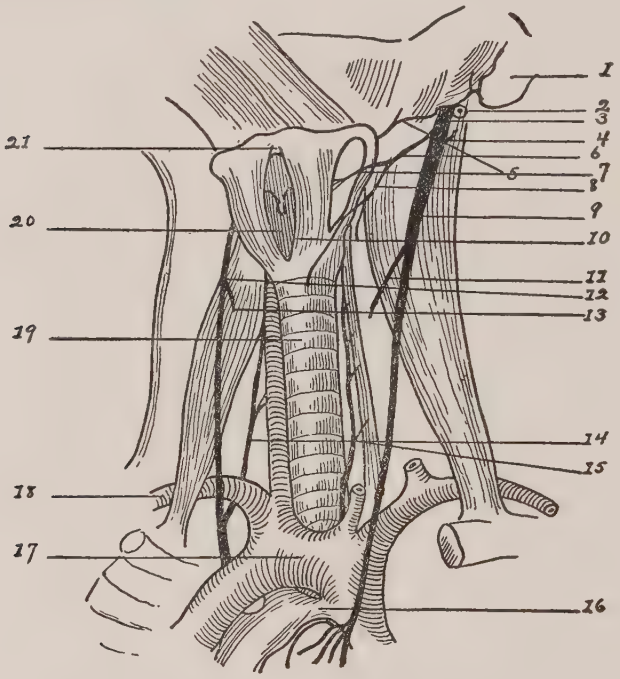

Fig. 72.—Relation of the pneumogastric and laryngeal nerves: 1, Mastoid apophysis; 2, jugular vein; 3, ganglionic plexus of the pneumogastric; 4, internal branch of the spinal; 5, pharyngeal branch of the pneumogastric, anastomosing with the pharyngeal branch of the glossopharyngeal; 6, superior laryngeal nerve; 7 and 8, internal and external branches of the superior laryngeal; 9, left pneumogastric; 10, thyrohyoid muscle; 11, cardiac branch of pneumogastric; 12, right pneumogastric; 13, right cardiac branches; 14 and 15, right and left recurrents; 16, course of the vein; 17, arch of the aorta; 18, right subclavian artery; 19, trachea; 20, thyroid cartilage; 21, hyoid bone (after Ziemssen and Henle).

The external branches of the superior laryngeal nerves enter the muscles so high that only the largest lobes can exert direct pressure on them. The motor impulses of all the intrinsic muscles of the larynx except the cricothyroid are supplied by the re-

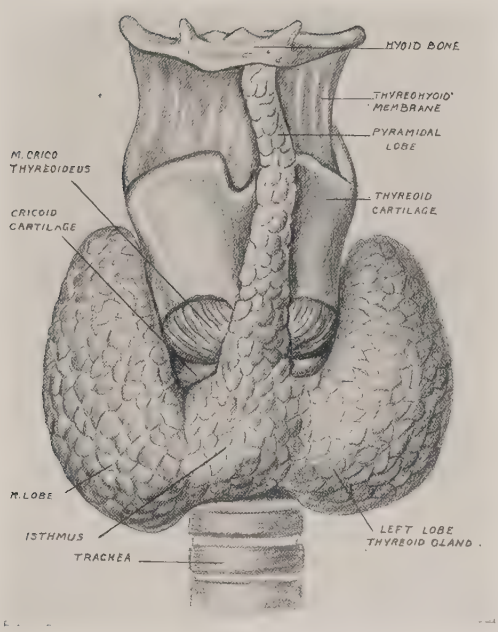

Fig. 73.—Showing relation of the thyroid gland to trachea and larynx (from Atlas of Anatomy, Sobotta-McMurrich).

current laryngeal; it also provides sensory impulses to the trachea and the mucosa of the larynx below the cords.

The superior laryngeal is the motor nerve for one intrinsic muscle of the larynx, the cricothyroid, the tensor of the cords, which modulates the voice; it also supplies the motor impulse to the depressors of the epiglottis, and the sensory impulse for the parts above the vocal cords.

The mechanism of the laryngeal muscles during phonation is exceedingly complicated. Briefly, however, we may distinguish three main movements: (1) Tension of the cords; (2) adduction of the cords; (3) abduction of the cords.

In regard to the character and frequency of the paralyses encountered in goiter cases, Matthews' observations, previously alluded to, are interesting. In his 289 cases of paralysis he noted —"total paralysis of the right cord occurred 18 times; of the left, 30 times. Partial or complete abductor paralysis of the right occurred 50 times; of the left, 83 times. Partial loss of both motions of the right occurred 38 times; of the left, 62 times. Bilateral tensor paralysis occurred in 4 cases."

Notwithstanding the fact that the recurrent laryngeal nerve supplies both the abductors and the adductors of the larynx, clinical observation demonstrates that the abductors are usually the first to become affected as a result of pressure on this nerve. This is known as Semon's law.

The symptoms of compression of the laryngeal nerves vary greatly, and in a general sense may be said to depend upon the size, degree, direction, character, location, and persistence of the disturbing element.

As these factors vary, making pressure on the several adjacent structures,—involving nerve-fibers singly, in groups, or the entire nerve-trunk,—so, too, may the symptoms vary and range from no perceptible voice alteration to complete aphonia; from no difficulty experienced in swallowing to grave dysphagia, and from entire absence of respiratory difficulty to extreme dyspnea requiring tracheotomy.

Matthews found that there was an increase of the paretic conditions as the size of the lateral lobes of the thyroid increased,

but that even great enlargement of the medium lobe alone was seldom associated with affections of the cord. This is, as might be expected, when the intimate relation of the lateral lobes to the recurrents is recalled and compared with the absence of this close relationship which the isthmus exhibits.

The position of the lobes, he also found, was equally important; for instance, in goiters of the same size he observed cord affections much more frequent in those cases with lobes located near the clavicle, especially if all or a portion of the goiter is intrathoracic. Hard lobes, likewise, are more apt to injure the nerves than larger soft ones.

The course of the paralyses is influenced by the vagaries of the etiologic factor. The sequence of events in compression lesions is, at the outset, phenomena of excitation and contraction, more or less transitory (spasmodic cough, aphonia, etc.); next, as the nerve filaments are more compressed, progressive motor paralyses as well as atrophy of the muscles innervated by the affected nerve, take place.

The history of Dr. Murphy's case and the clinical findings she presents emphasizes the value of a laryngoscopic examination. As the case well illustrates, the involvement of the vocal cords, due to the pressure of the thyroid, may be a deciding factor in instituting an operative procedure to remove this pressure. The prognosis in the case before us as to the restoration of vocal function is good, in view of Dr. Murphy's decision to remove the goiter—the source of irritation from which she suffers.

As a routine measure it cannot be too strongly urged that the larynx should always be interrogated in cases of goiter. In this connection it is not irrelevant to emphasize that, while the size of the goiter usually bears a direct ratio to the degree and severity of the laryngeal involvement, yet it must be kept in view that the direction, character, persistence, and location of the pressure are equally important, and that, not infrequently, an apparently small goiter, viewed externally, may be the unsuspected cause of irritations to the laryngeal nerves.

This latter type of what might properly be termed cases of "small-externally" goiter may, in their incipient periods of de-

velopment, for a considerable time, cause periodic, or more or less constant, manifestations of laryngeal irritation, such as chronic cough, hoarseness, etc., and these symptoms be explained on the basis that the patient is a victim of colds or laryngitis. A case under observation at the present time is of this character. A laryngoscopic examination may, moreover, by disclosing an unsuspected beginning involvement of the vocal cords, explain a later postoperative paralysis, thought to be due to the operative procedure, and, in addition, may prove the determining factor in instituting operative interference before distressing or irreparable damage has developed.

Dr. Murphy: With that history, you can exclude acute inflammatory conditions that occur in the larynx, as the patient states that she had no evidence of infection. The question that arises is, Was the cough the cause of the paralysis, or was the paralysis the cause of the cough? Did she have a lesion involving her vocal cords, such as a hemorrhage, or was that irritation of the recurrent laryngeal nerve responsible for her cough? or, when she coughed severely, did it cause hemorrhage into the capsule or into the gland, or whatever the condition may be that produced it, and was it a sequence or an etiologic factor? That is impossible for me to say, and it will not be possible for me to say even after I have made the dissection.

We will make the usual collar skin incision, cutting through the superficial and deep fascia and the platysma. I am elevating the platysma so as to expose the sternohyoid group of muscles. There is none of the usual hemorrhage which one sees in the exophthalmic goiter cases. There is none of that hemorrhage which we see coming from the skin incision in cases where we ligate the superior pole of the gland. The sternohyoid group of muscles is next separated in the midline and divided laterally. These muscles are exceptionally well developed. Their bellies stand out just as shown in an anatomic plate. Elevate them and divide them transversely in such a manner that they will be in readiness for suture subsequently. We must use every precaution here so as not to endanger the recurrent laryngeal nerve. The gland itself is now in the field. It is enlarged con-

siderably, and appears to be quite adherent to the surrounding structures. The vessels in the true capsule of the gland stand out very plainly. The most essential and important thing when operating on a goiter is to have plenty of artery forceps, and you want them in the right place at the right time.

I will free this gland from the outer side, so as to roll it up on the trachea. I am merely steadying the muscles. That allows the gland to come forward easily. With the dissecting scissors of Mayo we set free all the neighboring structures to which the gland has become adherent. The upper pole of the gland passes away around in the neighborhood of the esophagus. The gland is very much larger on the posterior border than one would anticipate from looking at it from the front. The bar or isthmus is also enlarged to such a degree that it was pressing on the trachea. The posterior lobe is larger than all the anterior portion. I have loosened it so that I can bring it forward. We are not removing the capsule of the gland, but enucleating it from behind forward. There is the superior thyroid artery turning upward as it came around from the inner side. There is a parathyroid. I will wipe that off from this capsule so as to be sure that it is not disturbed, and we will commence to go down to the posterior inferior portion of gland that is embedded deep. I expose the artery on the under side. There is the vein. It has a minus pressure on inspiration. You will doubtless remember that some years ago Crile suggested that we leave a portion of the thyroid on the posterior capsule, so as to be sure that we have not cut or injured the recurrent laryngeal nerve. I will peel this gland out of the capsule at the lower angle. The bar that projected down under the sternum is now visible, also the trachea. It is coming out nicely. Here is the isthmus that passes across the trachea. We are in the zone of the opposite recurrent laryngeal nerve. We will take off this isthmus and a small portion of this right lobe. Early in my experience I was called suddenly one morning to see a girl who was choking to death. The location of this isthmus and its firmness made me think of that case. The girl was cyanotic and scarcely breathing. I found that she had a sudden enlargement of her thyroid.

She was unconscious. I had my pocket case with me and I cut into her trachea and divided the isthmus of her thyroid, which was constricting the trachea and choking her. After this she began to breathe regularly, and subsequently the goiter disappeared completely. That case is reported in the literature, and in the report I called attention to the fact that the goiter disappeared after division of the isthmus. We never were able to satisfy ourselves as to the reason for it, but the clinical fact remained that it did.

We will place a clamp on the right lobe. In this procedure there is practically no bleeding if one keeps on the line of cleavage. In operating on an exophthalmic goiter case we have no occasion to get into a bloody zone. It is very important to be on the true capsule in making this excision, and the work has to be done in a slow and careful manner in order to obtain good results. Here is another one of the parathyroids at the lower angle. We will now excise this portion of the gland, which we believe will be sufficient to give this patient relief from her symptoms.

We will make some sections of the gland to see if we can account for its sudden enlargement, and then we will have a careful microscopic examination made. The muscles which were cut will now be reunited with catgut. The wound will then be closed with deep catgut and superficial horsehair sutures, so as to secure accurate approximation, layer for layer. A small rubber tissue drain will be inserted and allowed to remain for forty-eight hours, or longer, if necessary.

In analyzing these thyroid cases you can make the subject of thyroid enlargement and thyroid management very simple. It is not at all complicated. If you go back to the embryologic or histologic elements of which the thyroid is made up for your starting-point, in all the lesions you will find that the thyroid is made up, first, of essential, specific, secreting epithelial cells which are arranged in tubules and acini on the cluster order. These columnar cells rest on a basement-membrane. Connective tissue forms the supporting framework of these acini. There is where you get the enormous percentage of lesions—in these

cells and acini. Second, the thyroid also contains a large quantity of connective tissue which holds the gland together. If you could destroy all the connective tissue, the gland would look like a miniature cluster of grapes, the stems, however, being absent. The connective tissue binds the various parts of the gland together, and then there is, in addition to these two, the vascular supply, venous, arterial, and lymphatic, with no specific ducts leading from the gland, as, for instance, the chyle channels, to carry off the secretion. The secretion is taken up by the blood and not carried away by a duct. The gland, therefore, is made up of the epithelial elements in the acini of the gland, the connective-tissue framework, and vessels. Embryologically the epithelial elements are of what origin? The epithelial cells are of endodermic origin. The connective tissue and the vessels are both produced from the mesoderm. Malignant lesions of the thyroid are of two types only—one, the type that arises from the epithelial elements and is called carcinoma; the other, the type that arises from the connective-tissue elements and is called a sarcoma.

Let us go back to the most essential thing. Of the lesions that involve the connective tissue and the lymph-spaces between the connective tissue, you have what is known as metastatic infection in the gland, associated with grip, with typhoid, with scarlet fever, tonsillitis, with any of the severe infectious diseases. A suppurative tonsillitis may cause a metastatic infection in the thyroid, in which we believe the connective tissue plays an all-important rôle, and in which there is a hyperplasia of the connective tissue. In the acute stage the gland is enlarged and sensitive. There is elevation of temperature, and the patient is often delirious. The condition subsides slowly, and the swelling of the gland recedes. In the acute stage there are none of the intoxication symptoms that are associated with Graves' disease, the excessive production of thyroid secretion, or the excessive absorption of the thyroid secretion. When the inflammation in the gland subsides, exactly the same tissue changes occur as in the infections of any other loosely arranged glandular structure. There is cicatricial contraction of an

excessive quantity of connective tissue, which, in turn, causes strangulation and compression of the specific thyroid cells. Such compression may result in complete destruction of their secreting power, so that after a patient convalesces from the acute metastatic infection of the thyroid she may develop typical myxedema.

The following is a case in point: The patient had tonsillitis. I treated her years ago for an enlarged thyroid. Later, after she married, she developed some mild symptoms of Graves' disease. Under arsenical treatment the condition entirely disappeared. About three years after this she was confined, and a year after confinement she had a severe sudden tonsillitis, with some soreness in the lower part of the neck. She had metastasis in her kidneys, and an intense toxic nephritis, with blood in the urine, for weeks, blindness, convulsions, and finally unconsciousness. She was relieved by blood-letting by her husband, who was a doctor. After he got her out of that spell she recovered. Her kidneys began to improve, and her edema, which was extreme in degree, because she was a stout, large woman, gradually commenced to subside. She went to a southern resort to spend the winter, and got down to 180 or 190 pounds in weight, when her edema began to get worse again. She began to gain in weight. This was in November. I saw her in the first week of the following March. At that time her urine contained a large quantity of albumin, but a relatively small number of casts. There was no blood in the urine. She had weekly or ten-day explosions, in which her abdomen became enormously distended, passed large quantities of gas, had a severe headache, with a temperature of 103° to 105° F., but more frequently 103° F. Gradually the temperature would come down to normal and stay there until she got another explosion. She looked like a large mass of blubber, filling three-quarters of the bed, and weighing about 300 pounds.

What was the matter with her? The microscopic findings in the urine did not justify the conclusion that all that edema was from her Bright's disease. Looking her over, I wondered what had become of the thyroid gland, that always had been enlarged. Examination revealed that the gland was not pal-

pable. I concluded that it was probably a case of myxedema. Three-quarters of the hair on her eyebrows had disappeared; the hair in her axillary and suprapubic regions was gone. This confirmed the diagnosis of myxedema following contraction of the gland, the result of infection. We put her on sheep's thyroid and iodothyrin, and she fell in weight from 300 pounds to 120 pounds. She went to California the following winter, and developed an acute septic endocarditis, from which she died. She had always had a little albumin in the urine, but otherwise she was practically well. I cite that case merely to accentuate the contraction that takes place following infections of the connective tissue, and this means a destruction of the secreting cells of the gland. The nephritis complicated the case and made the differential diagnosis difficult.

I had another case that came from Nebraska during an acute attack. I saw the patient in a subsequent attack, in which the gland had contracted. She was fed on artificial thyroid.

In a third case of tumor of the tongue a remnant of the thyroglossal duct, which is embryologically the duct from which the thyroid gland is developed, and which opens on the surface of the tongue at the junction of the anterior four-fifths with the posterior one-fifth, the foramen cæcum. The moment I cut it open I recognized it at once as a colloid thyroid—an aberrant thyroid. I looked for another gland, but there was none. In the development of this thyroglossal duct the development of the thyroid proceeded downward to a limited degree and was arrested in the tongue end of the duct. The woman subsequently developed symptoms of hypothyroidism.

We began feeding her on sheep's thyroid, and kept it up for a year. Whatever remnant of the gland was left began to increase in size until finally she developed enough thyroid cells to carry her along without the thyroid we had been giving her. All symptoms of deficiency of thyroid secretion disappeared.

I cite that case that you may keep in mind the lesions which occur in connection with the connective tissue from cicatricial contraction, just as in the contractile form of cirrhosis of the liver. Connective tissue does the mischief.

Those are the principal lesions that occur in the connective-tissue elements. Now, as to the vascular lesions. There are lesions that occur in the blood-vessels, namely, dilatation and hyperplasia of all the vascular elements in the thyroid, and the result is a large pulsating thyroid without any symptoms of hyperthyroidism, without any symptoms of acute Graves' disease. There is no evidence of excessive production of the secreting specific, cuboid epithelial cells at all, but a great vascular pulsating mass, which bleeds severely when it is removed, but which is not associated with any of the dangers incident to removal of the Graves' type of gland.

The final and most important tissues in lesions of the thyroid are the acini and the cells that line the gland-lobules, that is, lesions of the epithelial cells and their basement-membrane. What are they? The normal product of the cuboid cells of the thyroid is what? Colloid material, and that colloid is produced and absorbed by a definite, automatic regulating system. When it is produced in excess of its absorption you have developed in the gland, if it is diffuse, large colloid masses, sometimes as large as a filbert, often larger, the typical colloid goiter, without any symptoms of Graves' disease. Then there may occur hyperplasia of the cells alone, with a perfect balance of absorption and secretion. When it is a circumscribed hyperplasia, you have an encapsulated adenoma—merely normal gland tissue. You often see this and can pick it out. Then you often have, in addition to that, excessive secretion within a distinct tubule, with encapsulation and retention of the secretion. That increases in amount and flattens the cuboid cells, making them squamous or flat cells. That changes the secretion from a colloid material to a serous material, and you have the development of serous cysts which often attain a large size. Again, in addition to this, there may be hyperplasia of the cells, a hyperplasia of the connective tissue, and the production and absorption of an excessive quantity of thyroid secretion, as we interpret it, with the symptoms of Graves' disease. We do not believe that it is a perverted thyroid secretion. We believe it is a normal thyroid secretion in excess of that used normally in metabolism, because you can

produce the condition artificially, as in the treatment of adiposus dolorosa with thyroid extract, which is a specific for it. You can develop Graves' disease nowadays, all except the exophthalmos. You can get the tremor and tachycardia, and all that goes with it, by the feeding of an excessive quantity of thyroid. Is it a hyperthyroidism? Let the pathologist and the internist quarrel about that. For our purpose, excessive gland tissue and a sequential intoxication are produced.

There is, then, left only the other lesion of the epithelial cells, a malignant lesion—a carcinoma. All these conditions lead right back to the essential action of what? To the cuboid cells in their relation to secretion, to absorption, and to their multiplication as the result of some stimulus which we know nothing about. So you see, it is not at all complicated now. There are three sets of lesions. They occur singly, circumscribed, and multiple, depending upon how much gland is involved. In the management of these cases you have all these conditions to consider. If you have a vascular thyroid, with pulsation in the beginning, it is a case, except in the very young girl, that requires the removal of a large portion of the gland. In cases of ordinary hypertrophy of puberty, if you put the patient to bed, apply an ice-bag locally, and administer arsenic internally, the hypertrophy, in the enormous percentage of cases, disappears without any surgical treatment whatever, not only in one case, but in hundreds of cases, and many more patients than that, in our experience, have not needed an operation at all. If there is present in the gland a circumscribed cyst as large as an apple, a hen's egg, or an orange, and you put the patient to bed, apply an ice-bag to the neck, and give arsenic internally, no good will come of it. You might as well give anything else internally or externally—it would have the same effect. Our line of treatment must be individualized. The personal equation and the differential diagnosis must be considered and carefully weighed before you decide on what treatment you will adopt in a given case.

In the exophthalmic type of goiter medical treatment is in nearly all cases a failure, except in those cases occurring in young

girls, and very young girls at that. The condition, as a rule, is a progressive one. Medical treatment at best gives only a temporary improvement. These cases ought to go to the surgeon. Therefore, if you make a diagnosis of beginning exophthalmic goiter, when there are present tremor and nervousness and tachycardia which come early, all these preceding the enlargement of the gland and the exophthalmos, that is the time to remove the thyroid, because then it can be done practically without danger. You avoid a long period of invalidism, you avoid the fatal conditions that otherwise would develop later, and which inhibit or prohibit operation.

Occasionally a patient comes in with symptoms of exophthalmic goiter, such as the tachycardia and the exophthalmos, but no goiter. I have had three cases of that type. There was no goiter, but they were said to have Graves' disease. Where is the goiter? Where is the thyroid gland? In the chest. In every one of these cases I was able to demonstrate the gland in the chest by percussion, and subsequently by the skiagram and fluoroscope. This is the vascular type of goiter. It throws an additional shadow in the upper chest, which is apparent in the skiagram under ordinary circumstances. The gland is always there. Sometimes there are manifested pronounced symptoms of exophthalmic goiter with a very small gland. Bear that in mind. Remember, it is not the quantity of thyroid secretion that is absorbed, nor the gland that secretes the most thyroid, that causes the most severe symptoms. It is the lack or loss of balance between the requirement of the body for, and the production of, thyroid secretion. Just as soon as that balance is lost, exophthalmic goiter develops, regardless of the size of the thyroid.

In the management of a case like this you go back to the management of the acute infection. You prescribe an ice-bag locally and rest in bed. An acute infection of the vascular type, if excessive, must be treated surgically.

Then we come to the colloid type. Once the gland attains a considerable size and is fixed, the patients do not, except young girls, as a rule, do very well under medical treatment, and the removal of the gland is required. There are scattered through-

out the gland masses of encapsulated colloid material as large as a filbert, which are not absorbed. Occasionally the gland enlarges synchronously from two conditions. First, growth and development of the epithelial elements in the acini, with the formation of new atypical acini, and, second, a malignant lesion may develop in another zone; then you have all the dangers that go with the malignant lesions of the thyroid.

The management of the hyperplasias of the thyroid in young girls is always medical. When the gland becomes firm and hard, the case is a surgical one and without danger to the life of the patient.

In the management of cysts, and particularly cysts of the isthmus, or one or the other lobes of the gland, enucleation of the cyst without removal of the thyroid is the operation to be done. There is usually present with it a thyroid of normal size, which is not removed. Simply enucleate the cyst with its capsule. That is all there is to it.

Then there are those large swellings of the gland that occur deep down behind the sternocleidomastoid muscle, and occasionally under the sternoclavicular articulation, which may suddenly endanger the life of the patient. I recall a case of that kind occurring in a doctor in this city. He had a cystic goiter; suddenly it began to distress him. He rushed to the West Side Hospital, and Dr. A. H. Ferguson immediately incised it, put in a tube, and saved the patient's life. He had a mild infection following that, but the wound closed up, and the doctor remained well.

When there occurs a sudden swelling of both lobes, strangulation may take place because of constriction of the trachea by the isthmus. You make division of the isthmus in such a case, lift the trachea and isthmus out from the substernal space, and the patient is relieved.

Now, as to the operations for the removal of the gland in exophthalmic goiter. Here one must individualize in a most careful manner before deciding whether or not to operate. These patients come to us in all stages of the disease, from the mild or comparatively mild type to the very severe type, when the

patient dies the day after arrival in the hospital without having been touched. It is useless to send a case in this superlative degree of exophthalmic goiter to the surgeon. If it is a case in the superlative degree, keep the patient at home, in bed, flat on her back, put an ice-bag on the neck, and administer arsenic. Put the patient mentally and physically at rest, and if she can be saved at all, she can be saved in the comfort and mental ease of her own home. If you expose her to the dangers of transportation and the anxiety that she feels about having an operation, she will die shortly after she arrives at the hospital, before anything at all is done, or on the way thereto, as we have experienced, and as the Mayos have experienced very frequently. The best treatment for hyperthyroidism of the acute and virulent and dangerous type is absolute rest in bed, with an ice-bag on the thyroid. Nothing else compares in value to that. That has been well established by Kocher, by Crile, by Mayo, by Moynihan, and by all the men who have had large experience in these thyroid-gland lesions. When the patient comes out sufficiently from that acute condition, you can commence the milder operation of ligating the superior pole, which is always associated with more or less danger. Why? Just the mere fact that you are only going to ligate tells you why. Because the patient is in so bad a condition that you do not do anything else, and if you do that, you are likely to pay the price with a fatality. If you look at the statistics of ligating the superior pole of the thyroid, you will find that the mortality is greater from it than from removal of the gland. In a case in which we operated a few days ago, we ligated the superior pole under local anesthesia. We did nothing else. That night the patient's temperature was 103° F. and the pulse 148. Why? Because he was very sick before we operated—he was intoxicated with his hyperthyroidism, notwithstanding the fact that he was for two months on the ice-bag treatment and rest in bed. He almost died at one time from a bowel fermentation of a mild degree, but in a week he came back again to the condition he was in before. He now has an occlusion of some of his thyroid vessels, and a lessened secretion from his thyroid. Later we will ligate the other pole and secure

sufficient diminution in the arterial pressure to get some connective-tissue development and contraction of the thyroid, and after that we will have to prepare for the removal of the other half, if necessary. The ligation of both superior poles, if the patient survives the operation, is not without danger.

I operated on a patient in February. Following the ligation, the patient made a nice recovery, notwithstanding that her's was an extreme case. The gland commenced to get hard, and we let her go home. When I returned from a European trip in August her feet and hands had begun to swell. She had albumin in the urine. She was brought back to Chicago. The hair had disappeared from the axillæ and the symphysis pubis, and as a result of the ligation she had progressive thrombosis in her yet enormously large thyroid. It was still a large thyroid, but it was as hard as marble from the thrombosis. All the secreting cells in the gland had been obliterated by the thrombotic process,—just what we wished to secure in a minor degree,—but this condition reached the superlative degree, and before we could place her under artificial feeding of thyroid she died, showing that when you ligate successfully you are still not free from the danger of the thrombophlebitis destroying all the gland-cells. If one is on the alert, however, and begins artificial feeding early, he saves these patients, because the thyroid glands of children, dogs, cats, and all sorts of animals can be removed and the subsequent result controlled absolutely by thyroid feeding.

In the operations for exophthalmic goiter one must be cautious as to the time of operation. He must individualize his cases. If the heart muscle is so affected that the pulse ranges between 160 and 180 at the time of the operation, or for days preceding it, postpone the operation until this condition is improved. It does not matter to the surgeon whether the heart-cells or the Purkinje cells (Crile) are mostly damaged, clinically the fact remains that these patients do not withstand surgical attacks well, and they must not be operated on until they are prepared and ready for operation. We did not have to take that matter into consideration in this case at all. This patient had compression of her trachea. You saw me pulling up the gland from the

side of the trachea, where it was caught under her sternoclavicular junction and compressed the trachea, being followed by an acute edema or hemorrhage into the gland, and that developed the condition she had. We did not have to fear her heart muscle or her Purkinje cells. We are not afraid that when we go upstairs she will die if we talk to her, as some of these patients with acute Graves' disease do after operation.

With reference to metastases of this gland, when these patients have malignant neoplasms they metastasize in different places. They metastasize in the bone. If it is a sarcoma, the metastasis occurs in the long bones. If it is a carcinoma, it also occurs in the long bones. If it is a metastatic normal thyroid reproduction which occasionally occurs, one which is not malignant, it always occurs in the flat bones, as in the calvarium. If it appears on the skull, it is a clean-cut thyroid metastasis of normal histologic structure—it is not malignant.

The treatment of these conditions is obvious, and we will not discuss it at this time.

DOUBLE CERVICAL RIB

HISTORY

PATIENT is a female, aged thirty-nine, a teacher by profession, who enters the hospital on account of a dull, aching pain in head, which radiates down the right arm.

Family history negative. Has always been well with the exception of ordinary diseases of childhood.

Present Trouble.—Commenced in April, 1911. The patient states that the right half of the head became numb. This numbness was accompanied by entire loss of sensation. This condition slowly improved and entirely disappeared in two weeks; then the shoulderblade on the right side became painful at intervals, and there was a spot near the inferior angle of the scapula, 3 inches in diameter, which felt cold. At this time the arm became numb and she could barely feel any sensation when any one touched the hand or arm. This was particularly noticeable on the thumb and first finger. The numbness gradually disappeared, but the arm was weak, became easily fatigued, and was cold most of the time, especially during the night. The ache continued in the shoulder during this time. In August the arm lost its feeling of numbness. This was four months after the onset of the trouble; the circulation became better, and the pain in the shoulder disappeared. The thumb and the first finger are still numb. The sensation in the rest of the arm is normal. The arm and hand are inclined to feel hot at the present time.

The other arm has given no symptoms of pressure. There has been no swelling of the arm at any time.

COMMENTS AND OPERATION

This patient complained of pain in her right arm, and particularly in her index-finger and thumb, in the zone supplied by

the musculospiral and median nerves. She has consulted a number of physicians for the trouble, and they all informed her that it was a neuralgia or a neuritis, and without endeavoring to ascertain the etiologic factor in the neuritis, it was prescribed for. A careful examination of her supraclavicular space shows she has a prominence above the first rib, and that this prominence extends forward to the posterior margin of the scalenus medius muscle; over this prominence on each side can be felt the pulsating subclavian artery. The least compression of this artery suppresses the radial pulse. Pressure of any severity causes pain to extend down the nerve-trunks, and particularly to the thumb side of the hand and forearm. There have been no trophic changes in the muscles supplied by the musculospiral or the median. It is clearly demonstrable, even without the *x*-ray, that the neuritis in this case is due to the pressure of a right seventh cervical rib; that the pressure has not so far interfered with the left brachial plexus or artery.

In the clinical history of cervical ribs we have two distinct sets of symptoms, depending upon the structure which is most interfered with by the advancing end of the cervical rib. The first set of symptoms is referable to the compression of the subclavian artery against the scalenus anticus muscle by the end of the rib, and produces ischemia of the arm and forearm, particularly at night or when the patient is compelled to hold the arms in the tense position for any considerable length of time in the daily occupation. The second train of symptoms is due to compression or irritation of the trunks of the brachial plexus. These trunks are also pressed forward against the scalenus anticus muscle, which causes the neuritis or neuralgia of which most of these patients complain before they have the ischemic symptoms. While the condition is congenital, it rarely manifests itself in the way of irritation until after the twentieth year. It is most marked between the thirtieth and fortieth years. The ischemia can be so pronounced as to cause gangrene of the fingers and thumb, more commonly of the latter. It rarely ever produces a complete paralysis, but any of the group of muscles supplied by the brachial plexus may be affected.

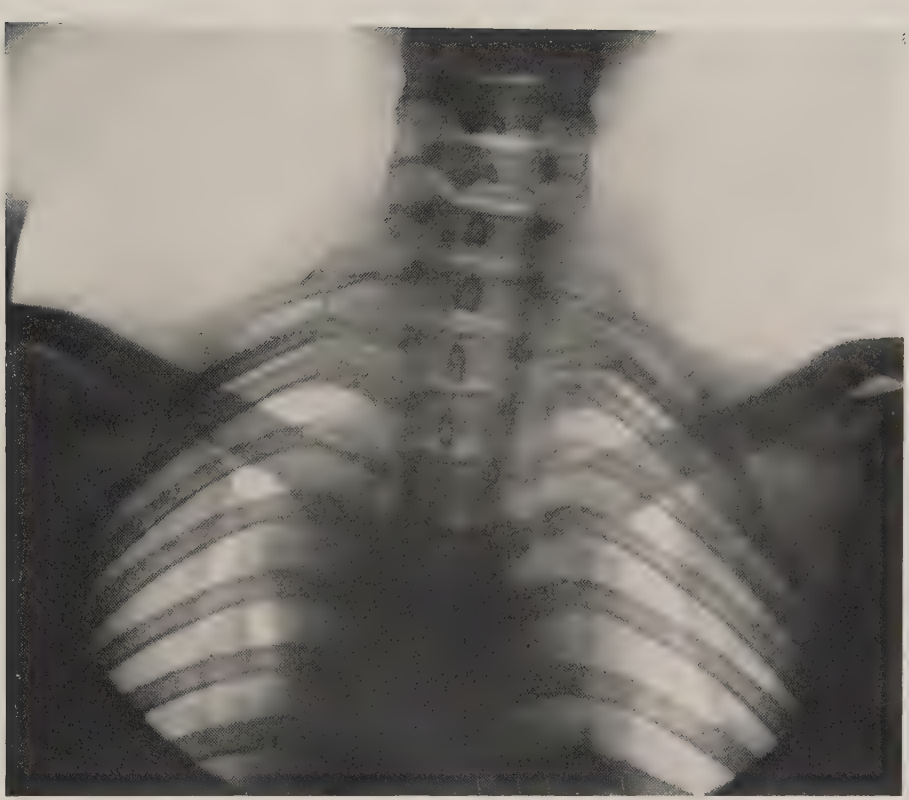

Fig. 74.—Skiagram showing double cervical rib.

The treatment of this lesion consists in the removal of the supernumerary seventh cervical rib. This should never be attacked from the apex, as on its apex are situated the nerve-trunks and the subclavian artery. The subclavian vein is never compressed by the supernumerary cervical rib, because the subclavian vein is situated anterior to the scalenus anticus muscle and cannot be caught and compressed by the advancing rib; it is displaced forward, but never to the degree of producing stasis in the vein. For that reason these cases are never associated with edema of the arm or forearm, and this accounts for failure of diagnosis of the disease in so many cases when really it should accentuate its probability. It can easily be attacked from its base by making an incision over and parallel with the anterior border of the scalenus medius muscle; one comes readily onto the neck of the seventh cervical rib; it is freed outward toward its tip, retracting the vessels and nerve-trunks inward. The neck of the rib is then divided with a short bone-cutting forceps, and the shaft grasped with a sequestrum forceps and the rib avulsed. The cartilaginous tip which remains attached to the first rib after the avulsion can easily be snipped off with a rat-toothed rongeur forceps. A deep muscle suture is made, and the skin is closed with horsehair. No drain. Subiodid of bismuth powder and a collodion dressing are then applied. The stitches will be removed in about ten days.

IMPACTED FRACTURE OF THE HEAD OF THE TIBIA WITH POSTERIOR LUXATION

HISTORY

School-girl, aged eighteen. Entered hospital February 14, 1912, because of old fracture of tibia and fibula with deformity. Family history negative. No past illnesses.

Present Trouble.—October 1, 1911, an express-train struck the automobile in which this girl was riding, killing two of its occupants outright, and throwing patient many feet. She was rendered unconscious. When next she remembered anything she was in bed. She was black and blue and sore from head to foot. The lower jaw was fractured, and the left side of head and face injured severely, but she apparently recovered completely from these injuries.

The right tibia and fibula suffered a crushing fracture without being compounded. She was in bed with a cast on this knee for seven weeks, and after that walked on crutches until Christmas, 1911. Since that time she has been able to walk, though with a limp, without the use of crutches.

COMMENTS AND OPERATION

Here is what has occurred: There was an impacted fracture of the external tuberosity of the tibia that split obliquely in a direction which permitted the occurrence of a luxation backward of the internal tuberosity, so that its anterior edge or margin of the internal condyle rests at the posterior margin of the external condyle of the femur. All the condyloid cavity of the internal tuberosity of the tibia is exposed behind the internal condyle of the popliteal space. Therefore there is an impacted fracture of the external tuberosity, with a rotation of the leg inward, so that the toe of the right leg, when standing erect,

points toward the arch of the left foot unless a voluntary effort is made to rotate it outward.

She has good motion in the knee, but when you flex leg at the knee beyond 80 degrees, the posterior edge of the internal tuberosity impinges against the femur. When she stands up, she toes in. If she makes an effort, she can throw her toes out, but in the normal position she toes in, because of the rotation inward. The accident occurred in October, 1911. Callus has formed. She has a fairly good joint. There is internal rotation of the leg, which is the most important impairment of its usefulness next to the backward luxation of the tibia.

The question arises, What are we going to do? We cannot bring these two tuberosities closely together because callus has filled that space. She has a movable joint. She has a joint that may be flexed almost to a right angle. We can divide the tibia below the line of fracture. If necessary, we can refracture the fibula and rotate that clear around into position without hazarding her joint one iota. We do not expose the joint by going into it. If we had this case originally, before callus had formed, we could have luxated the tibia back into position and put a nail clear across the surface, and thus have held it in position. We cannot do that now. The fracture has already united. We will take off the projection that is behind at the internal tuberosity, and which is limiting flexion, at a later operation, so that she will be able to flex her leg as well as she ever did. Remember, ideal surgery is that which produces results without hazard, locally or constitutionally. This is too good a joint to hazard now.

These skiagrams have great value in showing how important it is to make more than one skiagram, and how important it is to make the best skiagram possible. A skiagram is a part of the diagnosis now. It is a very essential part of the diagnosis. It is now a scientific proposition when you are doing this kind of work. Let me show you how important it is. There is here great subluxation of the tibia backward. The internal condyle is resting entirely external to the internal tuberosity. There is, you see, a very good joint. When she had the first picture taken

Fig. 75.—Before operation, showing deformity.

Fig. 76.—Skiagram of same case at different angle, showing lateral and posterior displacement.

Fig. 77.—Skiagram showing fracture of head of tibia, with impaction and posterior dislocation.

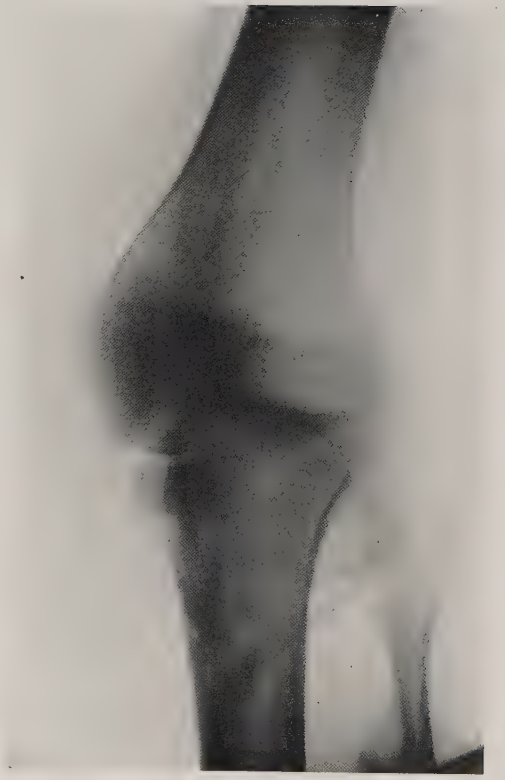

Fig. 78.—Skiagram showing apparent good position of bones.

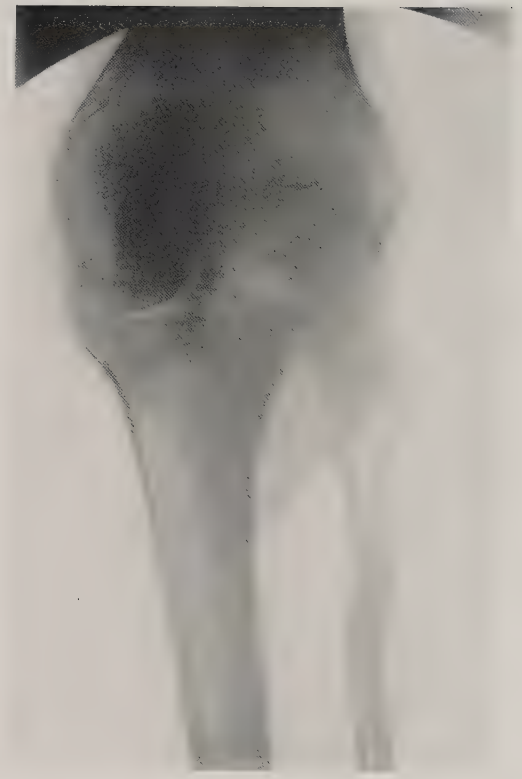

Fig. 79.—Skiagram showing apparent good position of bones.

Fig. 80.—Skiagram taken through the cast four weeks after operation, showing magnesium splint in position. No attempt was made to correct posterior displacement.

Fig. 81.—Skiagram taken through the cast four weeks after operation, showing perfect alinement.

one was led to say, "Why do you want to meddle with that bone?" But when you look at the two skiagrams made recently and synchronously, you can readily see how important it is to have these pictures taken in a scientific manner in order to get a proper estimate of the disturbance of relations. Let me show you where the greatest defect is. When you look at this joint from the side, you will see that the tibia is dislocated backward. You can bend the knee to a little more than a right angle. When you put the foot flat on the table, you see the foot is rotated inward and the leg is abducted, because it is shortened through the impaction on the outer side.

In this case we will have difficulty when we divide the tibia on account of the fibula, and on account of the external and backward luxation present.

There are some things about joints that we cannot yet control, and one of them is the fibrous type of arthritis following trauma, because it produces, first, a fibrous ankylosis and then a bony ankylosis. The fibrous type of arthritis is most common in the elbow, shoulder, and knee.

The tip of the tibial tubercle in this case is rotated inward, one inch from its normal location. What we want to do is to fracture the head of the tibia with chisel, make external rotation, and at the same time make abduction of the leg, and hold it in that position by means of a magnesium splint until we get bony union. We are going to fracture the tibia at about the place where it was fractured before—not in the same line, but on the same level, at its outer angle, because the same line would run up into the joint, and I am not going into the joint. The tubercle, the crest showing the line of division, the line of fracture, and here, as I elevate the periosteum, the line of luxation can plainly be seen.

I wish to cut across on about that level and make my division clear through the tibia, and rotate and abduct the leg. I will first cut the soft parts. This is the upper inner portion of the tibialis anticus. I am keeping far above the external popliteal nerve, so as not to interfere with its function at all. This is my landmark. You see, I have fractured the tibia just as I expected

to do, exactly in the line and in the way I predicted. We rotate the foot outward. See my landmark! I know just how much I have rotated the foot. I have the necessary adduction. Let me recapitulate: The line of fracture was exposed and a transverse division made of the tibia through to the line; after the fracture the limb was forcibly tipped inward to produce an elongation

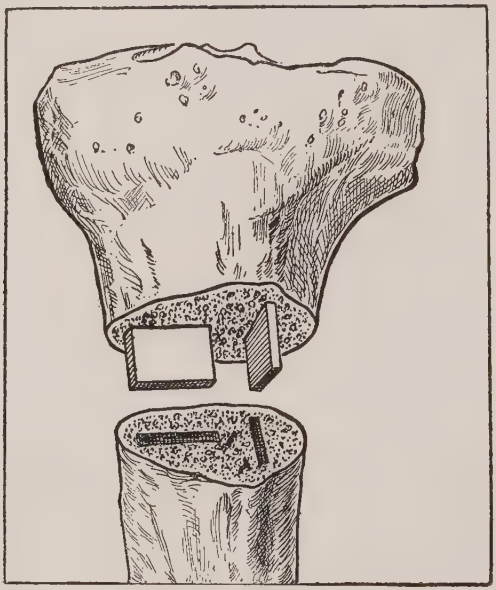

Fig. 81*a*.—Method of using magnesium ribbon splints in bones. Two splints in position in upper fragment with slots in lower fragment, into which splints will fit when proper approximation is made, thus maintaining separation and desired elongation as well as normal position.

of the fibular attachment. Rotation was then made of the tibia of just one inch—that is, external rotation of the lower portion of the tibia. When this was accomplished, the rotation was maintained by two magnesium interosseous splints, driven into the bone at acute angles to each other. These interosseous magnesium splints not only prevent a recurrence of the rotation, but aid in preventing the abduction of the limb; adduction will be controlled entirely by the plaster cast. When this union is complete, we will take off the posterior protruding internal

tuberosity of the tibia. There will be an excessive bowing backward of the leg in that position, but that excessive bowing does not matter so long as the line is plumb in her leg. That is the most important factor. You can see that the joint has not been hazarded at all. We are not in the joint, and the joint has not been disturbed. I am going to overcorrect the leg in the direction of adduction, because it will come back some on account of tension on the ligaments. Deep sutures of catgut and superficial horsehair sutures close the wound. No drainage. Subiodid of bismuth powder is applied freely; the line of incision is covered with gauze wet with collodion, and then a plaster-of-Paris cast is put on, extending from high up on the thigh down below the ankle. This cast will be worn for three or four weeks, and then a long sole-leather leg and thigh case, laced in front, will be worn for several months. The patient will not be allowed to put any weight on her leg for at least six or eight weeks. Passive motion will be instituted after two weeks.

I put the bandage on this leg at an acute angle, so as not to get any too great tension if the knee swells. That bandage can stretch without becoming tight. You should never put on a circular cast without immediately cutting it, no matter what the acute condition may be. If you do not cut it, you will pay the price by having atrophy of the muscles or a myositis and atrophy. You cannot foretell when it will come, but it will surely come. It is only a question of time. If the cast is cut, it is perfectly safe. The magnesium is always absorbed, but as it is absorbed hydrogen gas is liberated, and this must be removed by aspiration. A piece of magnesium ribbon one inch square and about one-eighth of an inch thick will liberate about 15 gallons of hydrogen gas. The absorption of the gas is not productive of any bad results except the inconvenience it may cause through tension. It may be removed quickly by aspiration.

Outcome of Case.—Complete primary union occurred. The magnesium splint was decomposed and gas formed, so that at the end of three weeks it was quite puffy. The gas was removed with an aspirating needle. At the end of eight weeks the patient

was permitted to put some weight on the leg. The limb was perfectly straight and the abduction of the foot was fully corrected. The skiagram shows the osteoporosis that occurred around the magnesium splint and also shows what appears to be a large granulation cavity. This is deceptive. It is the condition always present in fractures of cancellated bone tissue following union in the early stages.

TUMOR (HYPERNEPHROMA) OF THE KIDNEY

HISTORY

HOUSEWIFE, aged sixty. Entered hospital February 12, 1912, with hematuria as the presenting symptom. Family history negative as to malignancy and tuberculosis. She never has been associated with a tuberculous person, nor has she had any past illnesses. Menstrual history is normal. Menopause occurred at age of fifty, and she has not menstruated since. She has six living, healthy children.

Present Trouble.—In September, 1911,—five months ago,—patient began to have pain at times in the right lumbar and iliac regions. It bore no relation to the menses, which had stopped long before. It was sharp and aching in nature, and radiated down the thighs.

The urinations were accompanied by pain, and the amount has become increased, patient going to the urinal several times each night. In September, 1911, a small amount of blood was voided with urine for the first time. The amount was less than half a glassful. This, together with the pain, caused her to go to the doctor.

In November, 1911, in the middle of the night, she had a very severe attack of pain and vomited several times. In January, 1911, about the first of the month, she passed enough blood per urethra to fill a quart milk-bottle. Since then she has been so weak that she has stayed in bed continuously.

Her right iliac region, and just above and external to it, is the seat of great tenderness and pain. A tumor mass is present in this location. She had a chill one week before entering hospital, and cannot say as to the fever. Temperature normal on entering hospital. Examination of chest revealed a well-marked pigeon-breast. Heart was free from pathologic findings.

Cystoscopic examination February 13, 1912: "Very little congestion found about opening of right ureter, but a blood-clot could be seen escaping from it. No erosion or congestion of left ureteral opening. No sufficient evidence at right ureteral opening to justify a diagnosis of suppuration of kidney." Ureters were then catheterized, and 20 c.c. of urine obtained from each one. Urine from right side was much more cloudy. Laboratory findings as follows: Left kidney—numerous red cells present, 30 to 1/6 field; leukocytes, 6 or 7 per 1/6 field; a few granular casts present. Right kidney—erythrocytes, 12 to 16 per 1/6 field; leukocytes, 4 to 6 per 1/6 field. No casts found. Cultures showed presence of Staphylococcus albus. After repeated examination the tubercle bacillus was not found.

COMMENTS AND OPERATION

This woman first had blood appearing in the urine, and then she had severe colicky pains. She had nausea and vomiting, and the next day she passed, as she said, a sufficient quantity of blood to fill a quart vessel. You can see why she had nausea and vomiting. She had hemorrhage into the pelvis of the kidney, and had blood coming down through the ureter and distending it, and she had a reflex nausea and vomiting that comes with that condition. She has no history of having had chills; no history of fever; no history of having pus in the urine preceding these attacks of hemorrhage. Is that correct?

THE INTERNE: Yes, sir.

DR. MURPHY (resuming): You have here the history of these hemorrhages first as the principal symptom, and pain as a secondary symptom only. This is in a line of work that Dr. Lewis, of St. Louis, who is with us today, and whom you all know, is particularly interested, and who has done such splendid work that I would like to ask him to talk to you for a short time on the diagnosis of this class of lesions while I go ahead with the work. I have great pleasure in presenting to you Dr. Lewis, of St. Louis.

DR. BRANSFORD LEWIS, St. Louis, Mo.: Gentlemen: I feel that I am more or less trespassing to take up the time which

might be so much better occupied by Professor Murphy. However, I am willing to do what I can in the line of duty.

The diagnosis of genito-urinary affections is somewhat different from the diagnosis of a great many surgical conditions, in that the accessibility of the urinary organs to the general practitioner is less than is the accessibility of troubles with joints and extremities, the face, and so on. But a short step to the reaching of these genito-urinary organs has been made within the later periods of development of surgery by various means of mechanical diagnoses, such as the *x*-ray, the cystoscope, the sound, and various other instruments. All these things have really advanced genito-urinary surgery and put it on a tangible basis, to my mind. A man with the analytic mind of Dr. Murphy can analyze symptoms and do it much better than you and I, so that the mode of reaching a diagnosis is not so easily attainable for us as it is for him. We have to make use of materialistic means for reaching a diagnosis, and these means are the ones I have spoken of. A diagnosis means more than saying whether a kidney is affected or not; we want to know *how* it is affected. For instance, let us take the diagnosis of prostatic obstruction. To say that a person has prostatic obstruction is a very immaterial diagnosis. We want to know a number of things about prostatic obstruction: we want to know whether there is simply hypertrophy or contraction, or whether there is a stricture or a cyst formation. The exact form of the obstruction must be known, as well as the location of the obstruction and the condition of the allied organs, the kidney, and so forth. The same way with gonococcal infection. We must know the exact location of the infection, and each and every location must be differentiated. These things are all illustrated, not, however, in an analysis of the history so well as through the exact physical examination, and determining where these infections come from and the parts that are infected, and how extensive is the infection.

With reference to the history of these cases, I have often thought how unfortunate it is that these patients do not have pain in the early period of their infection. Just before coming here I saw a patient who had hematuric attacks which were of the

recurrent type for the last seven years, and this patient never had pain until the last month. The conclusion he reached was that it was necessary to consult a doctor and have his case analyzed, but he did not think it necessary to have this done until he got pain. Then, after seven years of trouble, he got pain and was willing to have himself investigated. This is an unfortunate feature in connection with these cases. They go through utter destruction of the kidney without having pain. I have seen the kidney with calculi in it absolutely destroyed without the patient ever having had pain—the kidney being absolutely destroyed with the absence of the symptom of pain. We must not, then, wait for the history of development of pain before we make a diagnosis in these cases.

I had one case recently in which the kidney was absolutely riddled with stones in a young man who was only twenty-six years of age. He had gone on for twenty years with bloody urine and a generally lowered condition of health. He never suffered very much from pain in his back. There were seven large stones in his kidney, occupying the kidney tissue from top to bottom, and without a history of pain. He had numerous diagnoses made, namely, general debility, gonococcic infection, and possibly tuberculosis, all of which diagnoses were incorrect, in that they were not based on the exact findings of the physical examination. In the examination of such cases one should demonstrate whether or not there is cloudy urine, which will lead one to examine the urinary organs from top to bottom. Examination with the cystoscope will show the presence or absence of trouble in the bladder; the ureters should be catheterized, and a determination made by means of the catheters as to whether the ureters are obstructed at any point, or whether there is a clear passage up to the kidney pelvis, drawing off the urine from each of the two kidneys, analyzing the respective urines from the kidneys, determining the rapidity of flow, as sometimes there may be one dram from one kidney and four drams from another. It is very essential to get an accurate analysis of the two urines from the kidneys.

Dr. Geraghty, of Baltimore, has lately developed an additional

mode of determining the functional activity of the kidney by means of the phenolsulphophthalein test, which is accessible to all of us. By injecting 1 c.c. of this solution and having two catheters in the two kidneys, in five, ten, or fifteen minutes there is the appearance of red urine in the kidney. If one kidney is diseased, that will be delayed, whereas the healthy kidney will show it in five minutes. If you take two urines the first hour of excretion from the two kidneys, they give a certain percentage from the normal, namely, 40 per cent., and the second hour, 20 or 30 per cent. There may be a rather low percentage of excretion in the first and second hours after the administration of the drug. If it runs to 40 or 50 per cent.,—and this test is quite accurate,—there is definite physical evidence when you get pus urine from one kidney and healthy urine from the other. The pus may contain colon bacilli or gonococci. The use of these various modes of physical examination is now just as accessible to all those who make use of them as the use of any mode of physical examination for any trouble in the human body.

Dr. Murphy (resuming): You see, now, I have lifted this tumor of the kidney out. I have it in my field. You can see, here is the neoplastic growth at the lower end of the kidney. Here is the stump of the normal kidney tissue. Here is the pelvis of the kidney. This kidney is just about the size which makes it the limit of comfortable removal from this position. There is a cyst on the upper side. I am going to take this out to bring the pelvis of the kidney out into the field or forward. I have been able to lift up the upper pole of the kidney, as you see, and expose the dilated ureter, as you see it here. I will free that ureter from its neighboring structures, catch the vessels around it, and, remember, the ureter is always back of the vessel. In the embryology of the kidney you know that the ureter is back of the vessel. There is an artery pulsating in front of me. I have my finger on it now. Those who are near enough can see it pulsating. I know just where I am. I am freeing the ureter as it passes around the artery here. I will grasp that now and get it ready for a separate ligation. The ureter is not infected, so far as we know. This puts it on the distal proximal portion of

the ureter. Now the ureter is divided. Here is the renal pelvis, and there are the renal vessels. We have all our field covered. I am trying this loose tissue which was attached to the kidney. We are down to the stump of this ureter. It has not been infected at all. It is perfectly dry. We have the whole thing in hand all the time. We tie the ureter. This tumor is either a hypernephroma or a sarcoma. It began in the medullary portion, and is almost confined to the medullary portion.

First, I will go through the technic of the operation, which I did not give, however, while Dr. Lewis was talking to you, and I take this occasion to express my appreciation to him for the discourse he gave you on the diagnosis and the significance of diagnosis in connection with tumors of the kidney.

The history of this case, you will notice, came along to the stage of hemorrhage. The stage of hemorrhage can occur in three lesions that are significant without having much pain at any time, but they have pain in varying degrees of early manifestations, dependent upon which one it is.

If you will begin with the stone type, these patients can have hemorrhage as an initial announcement of its presence without ever having had pain at all. But with the stone type the patient is much more likely to have pain and discomfort than with the two following types, which produce hemorrhage, and for that reason you expect it. But the stone may be large and impacted in the pelvis and never get to the stage of impaction in the ureter and give a typical colic.

The next type is tuberculosis. Tuberculosis can begin in the kidney, and the patient have only increased frequency of hemorrhage as a symptom of its presence.

The next type of tumor which produces hemorrhage without manifestation is the malignant type of neoplasm, and this is of the sarcomatous type, and in that we include the hypernephromata. These will go along for months and months without giving the symptom of pain, and finally the first indication of trouble you have is hemorrhage.

I want to show you this nerve. Look here! There is the nerve as it belongs there. As I pick it up I excite contraction

of the muscle. If you include the nerve in the suture, you cause the patient a great deal of pain. This is the most common cause of pain after suturing in the removal of the kidney or doing a nephrotomy. Do not cut it. If you cut it you will leave an area of anesthesia on the upper portion of the thigh. If you include it in the suture, you will give the patient severe pain a few weeks after its inclusion, so be careful that you see it in every case. I see it with each succeeding stitch, and I am avoiding it all the time. It is situated at the lower part of the muscle here, so that there is no occasion for including it in the suture. I am approximating the muscles; I am closing around the vein with double suture so as not to compress it. It is a continuous stitch, and put in so as to get muscular apposition—and good muscular apposition should be carefully attended to just as one cares for the opening in the anterior portion of the abdomen. Remember that a hernia can occur here; that when the kidney is permitted to remain, you can have a hernia of the kidney there and a very annoying condition.

In doing the operation we first cut down anterior to the quadriceps lumborum, open the fascia as it comes over, expose the cortex of the kidney, and free the kidney anteriorly and posteriorly above and below. In this class of cases you retain the capsule of the kidney. Even in a case of old infection you will find it of great advantage to leave the capsule in the abdomen and to enucleate the kidney from its capsule. In that way you come down onto the pelvis of the kidney with very little trouble. Where you have a suppuration in a large cystic kidney you not infrequently have adhesions high up in the course of the diaphragm, and you have great difficulty in getting them free. By taking it out in the manner in which I have described you have very little difficulty in freeing it, but if you take it out with the capsule, you have the greatest difficulty, and you may have a profuse hemorrhage in freeing the adhesions above the suprarenal capsule. When you come to the pelvis of the kidney: when you have removed the true capsule, you must not endeavor to ligate the vessels while the kidney is in position. You must use a rectangular forceps, such as I am using here, and this has been

in use for many years. It was not used when I was an interne. After you have taken the kidney out without its capsule, having enucleated it within its capsule, you cut down to the pelvis and loosen its capsule outward from the pelvis of the kidney, and that leaves the vessels. You can put forceps in here, anteriorly and posteriorly to the pedicle, and you slip this onto the pedicle and locate it from below. You do the same from above, and then you cut off the kidney and can have the vessels exposed. You can go on and ligate the arteries, if you wish, or do as I have done so often, disconnect the handles of these special forceps and extract them and let this stay on for a few days, and by that time thrombosis has occurred and you have no trouble with the ligatures. It is one of the easiest of subcortical enucleations. When you take out the true capsule of the kidney with it you have little or no trouble in exposing the vessels; you are not in danger of tearing the vessels in elevating the kidney. If you do a subcapsular enucleation you can tear off the vessel and have a fierce or profuse hemorrhage.

In doing one of these operations and removing the sac of a suppurative cystic kidney which had burrowed inward along the renal vein to the vena cava, I had to make a large incision, and I was peeling the sac off from the vena cava and tore it. In looking at it I simply put on three hemostats, went on with my enucleation upward from that, and I did an overstitch of the vena cava. You do not have to ligate any of the large veins. A simple suture stops bleeding from any large vein. Remember, the larger veins have less blood-pressure, and many of them have a minus pressure on inspiration and a slight plus pressure in the vessels of any portion of the trunk. It is a different proposition where you injure an artery. I went on with my enucleation, completed it, and the patient had no unpleasant symptoms.

There are some things about taking out a kidney that I wish to refer to. In the first place, in a large kidney like this, where you have a tumor in the kidney, you are not permitted to put on these forceps for delivery. If you do, you press the tumor and force the contents of it into the veins and disseminate it through the system and develop a fatal condition for the patient.

The way in which we elevate the kidney is to free it all around above and below. After we have freed the kidney all around,—let this represent the kidney [indicating],—I take and put that down in to the lower pole, pass it in front of the vessels and under the pole, and bring it around posteriorly. You see what I have done. I have a network. That is what I call my sling elevation. You get around and you can easily lift it out into the wound without the slightest danger of lacerating vessels. It is of the greatest assistance where you are going to do an operation on the pelvis of the kidney to throw a strip around and lift it out. You do not traumatize the kidney at all. You do not disseminate the contents of the neoplasm through the veins as you do if you produce traction with the hands or with an instrument in effecting delivery.

The next thing is to free the ureter. The pelvis of the kidney is behind. The artery is divided in two branches. This point was brought out by Israel, the upper branch going around the pelvis, and another going behind the lower half of the renal pelvis. When that condition occurs, as you expose the pelvis you see the renal artery in the field. You cut it and ligate it and go on with the enucleation of the pelvis, bearing in mind that the large vein and large artery are always in front of the pelvis. Therefore, when you wish to do any work on the pelvis of the kidney you attack it from behind, not from in front.

In making a diagnosis in this case we had hemorrhage without pain. We had no evidence of calculus; we had no evidence of tuberculosis, and we had no evidence of a discharge of pus irritating the neck of the bladder. That was the principal thing that might lead us to conclude quickly that this was a neoplasm. If she had a tuberculosis, or if she had a calculus in the pelvis of the kidney along with an irritating discharge from the pelvis, it would have eroded the zone around the ureter. There is no zone of erosion around it, but merely a blood-clot protruding from it.

As we take up the specimen and analyze it we see that the tumor began either in the pelvis or in the cortical portion of the kidney; it looks like a hypernephroma, and I rather think it is.

It began as you see, and gradually compressed the cortical portion of the kidney, so that there is less secreting surface than one would expect.

I will make another incision through the tumor and see the color of the remaining portion of it. It is a sarcoma or hypernephroma, and not a carcinoma. The first symptom she had was blood. Then came the attack of pain, with a large quantity of blood, but hemorrhage was the first symptom. I should say, from the gross appearance of this yellow mass, that it is a hypernephroma. The saving of this kidney was out of question.

What is the immediate prognosis, so far as the danger to life is concerned. It would be about 5 per cent. of danger, because there is a considerable portion of the kidney that remains which is normal kidney tissue, and the danger in removing a kidney, as far as the suppression of urine is concerned, is in direct ratio to the degree of normal structure still remaining. If the patient is perfectly well and has a kidney removed, when the cortical substance has not been destroyed, as is occasionally done with division of the ureter, the danger is approximately 30 per cent. There is suppression of urine, throwing all the labor onto the other kidney. Every one believes now that I am placing that estimate too high. I am positive I am not, that is, where you have a practically normal kidney. Where you have a kidney that is destroyed to a shell, with a big abscess as large as a slop-bucket, the danger of that is about 2 per cent. Just think of the contrast! The more kidney there is destroyed by the disease, that is, the secreting substance of the kidney, the more favorable the removal of the kidney is, because the work is actually thrown onto the other kidney and the other kidney readily assumes it.

You doubtless remember the splendid series of experiments which were made with reference to excessive secretion from resection of the kidney. It has been clearly demonstrated that if you take out two-thirds of a dog's kidney or a cat's kidney, and finally all of it, the other kidney, within a limited period of time, approximates almost the exact weight of the combined kidneys. It takes on the labor without a particle of trouble if it is thrown onto it gradually, but where it is thrown on suddenly, then the

patient has edema and swelling of the kidney under a normal tense capsule, and that kidney stops secreting by a compression strangulation, the patient has suppression of urine, uremia, and a fatal termination. Occasionally you may tide a patient over by doing phlebotomy and extracting a large quantity of blood, which lowers the arterial tension, lessens the tension on the cells within the capsule, and gives the secretion an opportunity to escape.

If there is any place where capsulotomy would be indicated, it is in that class of cases, in the acute congestive lesions, or in the acute infarcts which completely occlude the artery on one side and stop secretion on one side. If you will divide the capsule and permit the kidney to secrete, you occasionally tide the patient over a few dangerous hours, when he or she, as the case may be, will have uremia and die.

The final prognosis in this case is entirely different from the immediate prognosis. The final prognosis, if it proves to be a sarcoma, is that the patient is eventually going to die from sarcoma in the other kidney. Many years ago we collected statistics of a large number of cases of sarcoma of the kidney; this was before the time we differentiated hypernephroma, and before the time we differentiated the small round-cell neoplasm which so closely resembles sarcoma, and that we had not, up to that time, classified. With these two exceptions every single case of removal of the kidney for sarcoma had, within a period of twenty-one months, gone on to a fatal termination or to the development of a perceptible tumor in the other kidney. I do not know what Dr. Lewis' experience has been, but my experience has corresponded with those facts. Wherever I have removed a sarcoma of the kidney there was always a return of the sarcoma in the other kidney. The hypernephromata occasionally give us a permanent cure, but not often. The hypernephromata metastasize not infrequently very early in the manifestation of the disease, and you have this metastasis in the lung, in the bone, and in the pleura before you have indications of its presence announcing itself sufficiently to call your attention to the lesion in the kidney.

I recall a very bitter lesson I had, after reading the article

of a Scotchman, in a child that had been under my care. In the case of this child I made a diagnosis of sarcoma of the kidney; the tumor grew slowly. For a year I did not see the child, but in the mean time the tumor had grown a little larger, and at the end of two years it was still larger, and in three years I put a large interrogation point after my diagnosis. In four years the interrogation point became larger, and in five years I concluded I was wrong. I did not take out the kidney because I felt it would not prolong the patient's life to do so. However, concluding that I had made an error in diagnosis and that the neoplasm was some other type than that of a sarcoma, I took the tumor out, and within six weeks the patient had a tumor larger than the original one, and inside of twelve months she was dead. As long as I let it alone the patient was doing apparently well, but the moment I touched it, it immediately started out and produced its metastatic and local recurrence. It was an endothelioma, and endothelioma is the other type of tumor in connection with the hypernephromata. We do not know exactly where to put these endotheliomata as yet. They have no place in the classification of tumors. From a practical standpoint, however, you can put them with the sarcomata, particularly where the endothelioma occurs in the renal structure; the tendency is to recurrence of the same type of tumor in the other side.

So in this case, finally, the prognosis is very unfavorable.

CHOLELITHIASIS

HISTORY

PATIENT first visited doctor on February 21, 1912, on account of severe pain in the right hypochondrium, radiating to the back and up to right shoulderblade. He stated that about two years previously he was seized suddenly with a similar pain, since which time he has had frequent attacks of varying duration and severity. The recent attack began on February 18th, and has persisted ever since in spite of large doses of morphin. He has never had any chill or fever and never noticed any jaundice or change in the color of the stools or of the urine. On February 21st he was apparently suffering greatly, but the physical examination was entirely negative, except for tenderness in the right hypochondrium on very deep palpation. There was neither rigidity nor a palpable mass. The urine obtained at that time was acid, pale straw color, specific gravity, 1020, contained neither albumin, sugar, nor bile. Microscopic specimen negative in the centrifugalized specimen for pus, casts, or blood.

Doctor was called again to see him February 23d, on account of a severe attack of pain which had disturbed his night's rest. The examination this time revealed a temperature of 99° F. and a well-localized tenderness in the region of the gall-bladder. A specimen of stool was negative for blood both microscopically and chemically to the guaiac, as well as to Myers', test. There was an abundance of meat-fibers, but no excess of fat or fatty acid crystals. A test-breakfast has not been obtained. The diagnosis has rested between cholelithiasis and duodenal ulcer.

February 23, 1912, leukocyte count 22,000. Coagulation time, 2 minutes 18 seconds. Temperature on entering hospital, 99° F.

COMMENTS AND OPERATION

Why do we operate on this patient this morning in his acute attack? He is not icteric. He has no elevation of temperature, but he has a leukocytosis of 22,000. What does this mean? It means that he has an infection; that there is absorption of septic products; that he is putting up a good fight against it, and that he has some acute retention in his right hypochondrium. That is why we operate today. He came to the hospital last night with a history running over a long period of time. He has had retention in his gall-bladder for eight days. He is still having pains. If there is obstruction by a stone, it is in the cystic duct. The stone has not gone into the common duct. If you are going to save him from graver dangers, you should open the gall-bladder now, before the stone is forced into the common duct, and before he gets infection in the common duct with a stone, because it was such cases of stone in the common duct with infection that gave Kehr a mortality of 90 odd per cent. You do not have to operate in the acute attack where the stone is in the common duct and icterus is present. Where there is no icterus the stone is not acutely impacted in the common duct, because the patient has no bile in the urine and presents no evidence of icterus. The man has no elevation of temperature, and unless you made a leukocyte count you would have no evidence of infection. The leukocyte count tells the story. It is an important factor in aiding you to arrive at a diagnosis in this class of cases, and in the appendiceal cases where you have but little elevation of temperature, say a temperature of 99¾° or 100° F., but a leukocyte count of from 18,000 to 28,000 or 30,000. In this case it is of real value in the way of differentiation; it is not so important in the other class of cases. Let us figure this case out.

This man's brother came to me two years ago after many weeks of suffering, with a large abscess in the right hypochondrium, and with rupture of his gall-bladder from gall-stones. I opened the right hypochondrium and drained the abscess, and removed a number of stones which were extracystic. I did a secondary operation later, removing some stones which escaped from the gall-bladder at the time, and removed the gall-bladder

some months after that. Why should I let this man go on to the superlative degree of pathology as his brother did if we can open, drain, and remove a calculus and thus obviate a pericholecystic abscess, which might develop subsequently?

In making a differentiation between lesions that occur in this position you have to consider, first, the gall-bladder; second, stones in the kidney; third, the appendix; fourth, acute infections of the head of the pancreas; fifth, acute ulcers of the duodenum and stomach. You can rule out acute ulcers of the duodenum and stomach because they rarely ever cause the severe type of pain you have in the gall-bladder infections. I recall one exceptional case, a policeman, who came in here a number of times reporting that he had had recurrent attacks of acute abdominal pain. Finally he came in on the first day of one attack, and one could see, from the location of the pain, that it could not be the gall-bladder that was at fault—it was the stomach. It was a stomach ulcer which would get well between the attacks, it being very much like the crises that occur in tabes, except that he was more sensitive to pressure in his epigastrium. Involvement of the urinary tract—a urinary calculus—is a condition you can easily differentiate by the fist percussion method which I have demonstrated here repeatedly. (See p. 459.) It is the best physical sign I know of in differentiating abdominal lesions, and it aids more frequently in making a differential diagnosis in the way of excluding or including the kidney in the lesions than any method I know of.

Choleliths result from an entirely different condition than that causing urinary calculi. A cholelith is the result always of infection in the gall-bladder. The bulk of material which goes to make up the cholelith is cholesterin. The cholesterin is not precipitated from the bile that has been carried into the gall-bladder, but it is the result of degeneration of the epithelial cells of the mucosa of the gall-bladder produced by the infection, and precipitated and crystallized in the gall-bladder.

I am making a long oblique incision, ¾ inch below the costal cartilage margin, over the rectus muscle, and down the linea semilunaris, making the incision entirely through the sheath of

the rectus muscle. The fibers are split and displaced and the peritoneum opened. Here is the gall-bladder exposed.

The gall-bladder is not ruptured. It does not look like a gall-bladder. It is pale pink, acutely infected and infiltrated, and looks like an inflamed stomach. This is a typical picture of a case of acute infection. Note the pale pink color instead of the normal slate color. It looks like a peach. I cannot even tell whether or not he has a stone, on account of the tension and non-compressibility of the gall-bladder, and I am going to put in a coffer-dam of gauze. I will take the tension off of his gall-bladder first by aspirating it. The first bile extracted is usually clear, as the pus is usually situated down in the bottom or in the cervix of the gall-bladder. We have now the tension off of the gall-bladder, so that we can palpate the neck of it. I find there is a large stone in the bottom of the gall-bladder. It has slipped by, and it feels as though it is impacted in the cervix, but it has not ulcerated through. With a little manipulation I am able to free it from this hour-glass pocket and press it back into the gall-bladder. We are getting ready to make closure of the gall-bladder before we permit this septic material to escape by placing two rows of catgut puckering stitches in its dome, ½ inch below the other. This stitch is going into the wall of the gall-bladder but not all the way through. I am going to make an entropion of the cut edge of the gall-bladder and drop it back into the free peritoneal cavity.

When I wrote my first article on gall-bladder surgery and the use of the button in that connection you could put all the recorded cases on a single page of ordinary paper. The button was originally intended for the operation of cholecystenterostomy, and was used primarily for that purpose. Now, of course, its use for that purpose is rather rare, since we have developed gall-bladder surgery. Kocher, in his latest work, says that while the button has disappeared to a certain extent from other fields of work, still, where you are unable to clear the duct and wish to do a cholecystenterostomy, the button holds a preëminent position. For posterior gastro-enterostomy we still use the oblong button

because we believe we can do the operation better with the button than with other means.

We will put in a second row of sutures outside the first, because the inflamed gall-bladder will not yield like the ordinary soft gall-bladder does. We have protected the field of operation by sponges, and we are now going to open the gall-bladder and remove the cholelith. So infiltrated is the gall-bladder wall that the opening does not contract. Here is a very pretty example of necrosis of the mucosa from the continued ischemia and pressure of the retained product under tension. Now comes the débris from the retained infection product. Every now and then a considerable quantity of pus comes out. We broke this stone in displacing it. That is the one that got down in the cervix fellæ first. The smaller ones could not pass by it. If they could have gotten by, he would have had recurrent attacks of jaundice.

We inject atropin in most all operations preceding the anesthesia, particularly in adults, as it stops secretion in the throat and, we believe, lessens the liability to inhalation pneumonia, because it does diminish the secretion so markedly. One of the objections made to its use was that it did not help the patient to expectorate. They do not expectorate when they are asleep, and I believe that argument against its use is not a valid one. There is no question in the minds of those who have used it for a considerable length of time that it does lessen the quantity of secretion. We are notorious faddists in medicine, and we reserve the right to change our opinions. For the present that is my opinion, namely, that it does lessen the secretion.

We have the gall-bladder closed around the tube and the tube secured to the gall-bladder. We will put in another stitch, making it additionally secure, and we are through. We will commence to remove the packing which we had around the gall-bladder and remove with it the gall-stone débris and infected material that rest on the sponges. We want another tube for the outside. This is what we call the extracystic tube for drainage of the peritoneum, which we make use of in all the cases in which we have suppuration of the gall-bladder. This tube drains the hepaticoduodenal fossa. At the time we first described

these operations we mentioned, first, the type of drainage and fixation to the anterior abdominal wall. Second, an ideal cholecystostomy, that of opening the gall-bladder, cleaning it out, closing it by a double row of sutures, and dropping it back into the peritoneal cavity. Third, opening the gall-bladder, draining it, securing a tube in it, and dropping it back into the peritoneal cavity. The latter process we denounced years ago, as we did not feel it was safe to drop the gall-bladder into the peritoneum in this position with a tube in it. Since that time the work of Mayo has emphasized that it is the safest plan, and we have adopted it. It is a plan that leads to very little trouble subsequent to the operation, as there is no fixation of the gall-bladder to the peritoneum. One of the conditions which we have, if we should fix the gall-bladder to the abdominal wall, as we did in our original operation, is the traction following the operation, which is painful and annoying and inconvenient to the patient, and we believe that is of sufficient moment to warrant one in doing the procedure which we are doing now, knowing, as we do, that adhesions form rapidly around the gall-bladder and rubber tube and that these adhesions protect the free peritoneal cavity.

The tube is left in three or four weeks, depending on the degree of infection. Here there will be considerable necrosis of the mucosa of the gall-bladder—it will come out in fragments, and we will keep this tube in for a longer period of time.

The reason he is having necrosis of his gall-bladder is because it was under tension in the first infection. I have seen the entire mucosa of the gall-bladder come away following infection. You could pick it right out after opening the gall-bladder. I recall my first case of this type. The patient had three attacks. He had gangrene of all coats of the gall-bladder, but the peritoneum was restored and the mucosa sloughed out and came away.

We have colon bacilli present here, and, therefore, we are in danger of having a colon bacillus infection of the cellular tissue. We mop the areolar tissue with a 1 per cent. solution of lysol, as it seems to have a good effect on colon bacillus contaminations.

In connection with gall-stones: 98 per cent. of the weight of all gall-stones is made up of cholesterin; over 99 per cent. of all

gall-stones originate in the gall-bladder; a fraction of 1 per cent. of gall-stones originate in the intrahepatic biliary channels. Many of the gall-stones found in the liver are transmitted to the liver from the ducts by having obstruction below caused by a large stone. Small stones following it into the common duct and not able to pass it are forced up into the hepatic duct and back into the liver. Ninety-nine per cent. of gall-stones originate in the gall-bladder, and always as the result of infection. The most common place, therefore, where we have disturbance from stones is in the gall-bladder; the next is in the cystic duct; the next, in the common duct.

It is estimated that about 10 per cent. of all adults have gall-stones. It is further estimated that only about one in ten of these has manifestations of the presence of stones. These symptoms consist in manifestations of the presence of a foreign body in one of the positions I have mentioned. When these remain in the gall-bladder undisturbed, the rule is by secondary infection that they give little or no trouble. As they develop they give trouble in many ways. First and foremost is the disturbance of their position, so that they are dislodged or displaced as they attempt to pass into the neck of the gall-bladder and out of the gall-bladder into the ducts. Second, disturbances are produced by the secondary deposit of infective material around the stones or on the surface of the gall-bladder, which is irritated by the stones. Third, disturbances are produced by the increase of secretion from this infection, the secretion forcing the stones out or carrying them out with it through the neck of the gall-bladder into the cystic duct. They are carried on from the cystic to the common duct and from the common duct into the intestine.

When they are not infected and impacted in the gall-bladder, the way in which they produce trouble is by their mechanical presence only. When they become impacted in the gall-bladder they produce manifestations by the irritation that they cause and the evidence of absorption from the infection that occurs in the gall-bladder. That is great or small, depending on whether or not the contents of the gall-bladder are held under high tension by the impaction of the stone in the cystic duct, as was the case here, or

whether it is merely an infection of the gall-bladder with virulent microörganisms, which is associated with chills and fever, edema, and swelling of the mucosa of the cystic duct, and makes the patient very ill, with the manifestations of infection, or whether it is an infection and ulceration of the neck of the gall-bladder from the impaction of a large stone, or retention in the neck of the gall-bladder without infection, gradually increasing the quantity of seromucous material in the gall-bladder under pressure, and producing continued distention of the gall-bladder. Whenever there is present only mild infection, and a stone of this magnitude is impacted in the neck of the gall-bladder, there is always a tendency to enlargement of the gall-bladder. The symptoms of cholelithiasis then are in this order: First, pain; second, nausea and vomiting; third, local sensitiveness in the region of the gall-bladder; fourth, elevation of temperature if infection is present; fifth, the presence of icterus when the stone has passed into the common duct—never before. You can see, therefore, that the element of icterus comes into play only when the stone is in the common duct. The order of symptoms is practically the same as in appendicitis, except that the sensitiveness is in an entirely different position. In making the test for sensitiveness perpendicular percussion takes precedence over others. Place the middle finger at right angles over the gall-bladder, and strike it with some force. Why is it that when we have so much pus in the gall-bladder, as we do from excessive infection, there is not a higher temperature? Because the fundus mucosa of all viscera has a meager supply of lymphatics. One rarely finds a temperature of 103° F. with septic cholecystitis, because there is so feeble a supply of lymphatics in the mucosa of the fundus of the bladder. That is an entirely different state of affairs from what exists when a stone passes into the cystic duct. There may be a small stone in the cystic duct, with retention and ulcer and pus present, with the temperature running up to 103° to 105° F. once or twice in the twenty-four hours, and continued for months, associated with chills, and resembling many of the types of ague, and still the patient may never have jaundice or a colic. The stone may pass on into the common duct,

and because of the presence of the stone in the common duct and the existence of infection there occur elevation of temperature, chills and fever, and icterus.

Why is it that there occur such pronounced elevation of temperature, chills, and fever with stone in the cystic duct and in the common duct, and so rarely high fever or chills when the infection is in the gall-bladder? In the latter case there is an enormous quantity of pus—even to half or three-quarters of a pint, while in the former, two-duct types one may find only three, four, or five drops of pus around the stone. The reason for that is that the stone has abraded a narrow channel rich with lymphatics. You have just the same conditions with the presence of chills and fever and infection with a stone there that you have when the stone is in the ureter, in the presence of infection, and that is why chills and fever are so common in these two particular positions, and that is why infection in this particular position is so dangerous.

Kehr, in the early history of gall-bladder work, did a large number of choledochotomies in the presence of icterus and infection. Kehr found that when he operated in the presence of chills and fever and jaundice practically all his patients died from the infection and the icterus associated with it. When he operated on patients who had chills and fever without icterus, and with the stone in the cystic duct, only a small percentage of the patients died; but when he operated on patients with a temperature of 102° to 103° F. with infection of the gall-bladder, he practically lost none of them. The three different positions in which gall-stones lodge and the nature of the attack led Kehr and others to avoid operating on patients in the acute infective stage in the presence of jaundice.

I was led to operate on this man today because he had no jaundice. He had a stone impacted in the gall-bladder cervix for eight or ten days, and it would be only a short time, if the stone is of moderate size, before it would reach the common duct, infection would take place, and the patient would be exposed to all the dangers that go with such a condition. I operated in the acute stage because I was able to differentiate that his infection

occurred in the gall-bladder, with a low temperature and a high leukocyte count, in contrast to what we would have had if the stone were down in the cystic duct or in the common duct, and in the absence of jaundice. We avoided the danger of perforation of the gall-bladder from that stone, which was impacted in the neck and might finally have ulcerated through, and the patient would have had a pericholecystic abscess which would hazard his life. Now, we have avoided the superlative degrees of pathology in this man's case and all the superlative dangers by a timely operation with a differential diagnosis as to where the lesion was.

The type of chills and fever occurring with small stones impacted in the cystic duct makes it difficult to diagnose in a differential way. Perhaps I can do no better here than to cite the history of one particular case which I saw in consultation. The man gave this history: He had had ague—chills and fever—for eleven years. He had been to all climates of the world to get rid of the ague. He was a wealthy man, and was able to go wherever he desired. He went to this place and to that place and finally landed in Pasadena, California. There he began having chills and fever of an irregular type. He had them at first once a day, then twice a day; then every second or third day. He went on in this way for some eight or ten weeks. He was losing ground all the time. He was seventy-eight or seventy-nine years of age. The chills were followed by a very high temperature—as high as 105.5° F. He never had a history of colic; he never had a history of jaundice; he had no history of trouble in the region of the gall-bladder. He had had innumerable examinations made for the plasmodium of malaria, and they were all negative. The only symptom he had was sensitiveness on deep palpation. You could hook the finger under his costal arch. He was a long, lank, lean Yankee, had no fat on him, and when he inspired with the finger hooked beneath the arch, it hurt him severely. The diagnosis was made of infection of the gall-bladder, and he was told that he should submit to an operation. He went to the hospital and I operated on him. He had a very small gall-bladder—not larger than the two distal segments of my finger. It was embedded in a large mass of adhesions. There was no stone in it.

It was a little pocket of pus surrounded by an enormous mass of cicatricial tissue. We split it open down into the cystic duct, but we could not put in a drain because the lumen was not big enough. We sewed a rubber tube with silk over the cut edge so as to prevent a closure of the opening and fixed it there. The next day he had a terrific chill, with a temperature of 106° F. He recovered and is alive today. The operation was done many years ago, and he has never had another chill. This was the last of this ague. Drainage was continued for several months.

I have cited that case to show you how high the temperature can be and how long the trouble can continue without the patient ever having a colic, without ever having a pain in the neighborhood of his gall-bladder. The only symptom in this case was sensitiveness. The temperature course was classic of cholecystic or cholangitic infection; that is, the temperature would go up to 104° to 106° F., remain at that point for a few hours, drop suddenly to normal, stay normal indefinitely, and do the same thing, up and down again, so that it was not the temperature-curve of sepsis, but the temperature "angle," which I described in Sajous' work when I wrote my article on gall-bladder infection. I described it as the temperature "angle" of cholangitic infection. It is classic in its appearance. The only instance in which you find it otherwise is in the infections which occur in infected erectile tissue or angiomata, as in the corpus spongiosum and corpus cavernosum, in the venous angiomata of children, and infections in the portal circulation.

We have come to recognize the fact in our modern work that where cholecystitis is caused by a cholelith, the disturbance is primarily in the gall-bladder. We are getting a smaller and smaller number of superlative degrees of pathology in cholelithiasis, and we are able to make a diagnosis, in a great percentage of cases, without the presence of jaundice at any time in the course of the disease. Years ago, in checking up 100 of our cases, we found that in only 14 of them did jaundice occur at any time in the history of the disease, so that you must make your diagnosis in 86 per cent. of the cases without the aid of jaundice.

When the diagnosis of cholelithiasis is made, then is the time

to operate. Do not postpone operation and hope that the case will go on without producing further trouble, because these cases go on until they produce a lesion that greatly or materially hazards the life of the patient. Therefore, immediate operation after the diagnosis is made, or immediately after the acute attack passes over, is the time of election for the operation, because it is a procedure of little danger to the patient.

An examination was made after two weeks with an electric cystoscope. It showed that a small stone was present in the hour-glass contraction zone, where the large stone had formerly been lodged. The cystoscope was pressed on to this to the round ligament; a hook passed through the cystoscope to the stone. The stone was rotated and jammed against the edge of the cystoscope, and by this means it was extracted from its position.

The drainage was removed at the end of five weeks. The gall-bladder mucosa had resumed a smooth, glistening appearance, in contrast to the trachomatous appearance which had been present at the time of operation. The smooth condition of the gall-bladder is our guide in removing the drainage-tube.

TYPHOID SPINE

HISTORY

PHYSICIAN, male, aged twenty-seven, single; has lived in the East all his life. Grandfather died of tuberculosis of lungs fifty years ago. One cousin has tuberculosis, but patient has never lived with him. No malignancy in the family. Two brothers and one sister living and well. Never had any venereal infection. Had measles at three years, following which had some trouble with both ears—a double otitis media. The left healed completely, but the right showed discharge intermittently until after the recovery from typhoid fever in December, 1910. Thus the chronic otitis media continued over a period of twenty-three years. An acute attack of right mastoid involvement at the age of twelve, and again after patient had been swimming during the summer of 1909 or 1910. Nasal diphtheria at age of eight years, which patient thinks aggravated the ear trouble. At age of fourteen had a fall, at which time it was thought skull was fractured at the base; there was a marked motor aphasia, which continued for three weeks, and some impediment in speech for two or three months; at the time of fall there was a copious bloody discharge from right ear.

Present Trouble.—Dates back to time when patient was seventeen years of age, during the time that he was still having the intermittent discharge from the right ear Patient noticed one day, while playing the piano, that he had pain in the metacarpophalangeal joint of forefinger of right hand. This was December 15th, and pain, "*redness*," and swelling did not disappear for about nine months, when the doctor immobilized the finger. *x*-ray showed thickening of the metacarpal bone under the periosteum. About five weeks after this finger trouble patient noticed, while skating, that "one" hip was sore on motion and

caused a limp when walking. No swelling, no chills, no fever. Patient was in bed about one week, and upon getting up pain in both heels appeared. A trip abroad and three weeks' treatment at the baths in Italy gave no relief from the "rheumatism."

Went to New York and was treated with orthopedic shoes by a surgeon; some improvement in heels. The doctor said it was "flat-foot." Treatment for a long time.

Four and a half years after onset pain appeared in both hips. Pain was severe when first beginning to walk or run, but "wore off" after exercise. For three years following the pain in heels and hips was marked only upon severe exercise. While serving in hospital, doing heavy lifting, etc., patient noticed a dull, aching pain in his back, most marked in the morning, and disappearing during the day. Leather jackets, braces, hip supports, etc., were advised by several doctors.

Typhoid fever December, 1910; sick four to six weeks. Positive Widal and positive blood culture. Spinal trouble was greatly aggravated by the fever, and patient was subsequently treated for typhoid spine. At present had no pain in heels nor fingers or any other joints. Spine is painful upon exertion, as are the sacroiliac joints. Has sensation of loose joints in hips, which is overcome by pressure on hips.

Examination February 27, 1912: Has pubic and suprapubic varicosities mostly on left, showing that the deep iliac veins are closed. This condition has existed since his typhoid fever. Thigh (left) larger and skin soft. Lumbar spine and sacrolumbar articular and cervical vertebræ are fixed. Edema in left leg. About 80 per cent. of spread of limbs when flexed at right angles.

Wassermann test: Wassermann serum reaction for syphilis negative, but there is enough inhibition to hemolysis to make the presence of some abnormal condition probable.

Dr. Charles L. Mix: We have here a case of typhoid spine which Dr. Murphy has asked me to show you this morning. This young man had typhoid fever some time ago. He is a physician here in attendance at Dr. Murphy's clinic. The patient's attack of typhoid fever was subsequently followed by a typhoid spine, which is probably due, as these cases usually are, to the residual

effects of the original infection. It is just as rational to suppose that the residual effects of the original infection can be aided by vaccine in a case of typhoid fever as it is to suppose that they can be aided in a case of streptococcic or staphylococcic infection. Consequently it has been thought wise to give him the benefit of vaccine treatment, and we are going, therefore, to give him this morning, subcutaneously in the arm, 500,000,000 dead typhoid bacilli. In ten days we will give him 1,000,000,000, and ten days after that another billion. If, at the end of three weeks' time, he still has further trouble, we will perhaps be justified in giving him a fourth dose of 2,000,000,000 bacteria.

DR. MURPHY: Will you give us the present status of typhoid vaccine as a prophylactic at this time?

DR. MIX: The results in the United States army have been exceedingly good in the prevention of typhoid fever by vaccination. I do not know whether you are familiar with the general subject or not. Vaccine prophylaxis for typhoid was first introduced extensively at the time of the Boer War. It met with a more or less indifferent reception, by reason of the fact that it was impossible for Sir A. E. Wright to follow up the end-results, so that at the end of the Boer War it was an open question as to whether there was real good done by it. It was abandoned temporarily. Subsequently, however, in England, about seven years ago, it was reintroduced by Sir W. B Leishman, and at that time it was used a good deal more successfully, especially in the English army in India, where the results obtained were very excellent. It then began to be experimented with in our own country. The first experiments of any note were made since 1908. In June, 1910, a small battalion of engineers stationed at Washington were ordered into the field. This group numbered about 120 men. Of them, over 90 had been vaccinated, and none of these acquired the disease, whereas of the 25 unvaccinated men, 6 subsequently developed typhoid fever. The last great experiment was made in the recent assembly of the army at San Antonio at the time of the Mexican rebellion, and on that occasion it was deemed advisable to make the matter a compulsory one, and each soldier who went to Texas was given the vaccine. The re-

sult was that only two or three cases developed in several thousands of the troops, showing conclusively that the prophylactic value of the vaccine is very great indeed.

I am very much interested in the general subject, as all internists are. Last September I developed typhoid fever myself, and thought I would try a little of the prophylaxis upon my own body. I went upon the theory that if the typhoid germs cannot be obtained from the blood until about five to six days after the onset of the fever—in other words, if it takes five or six days to bring about typhoid bacteremia, and if it takes only twenty-four hours or so to get the effect from the autogenous vaccine, it ought to be possible, in the first two or three days of the fever, to get ahead of the infection, the same as is done in the case of hydrophobia. The stage of incubation is sufficiently long in that disease to enable the physician to get ahead of the infection in the treatment of the patient. Why might it not also, in the very early cases of typhoid fever, be possible to get ahead of the infection? I, therefore, took a good-sized dose, 250,000,000, on the third day of the fever. The result was very interesting. I ran through a total number of twenty-one days of typhoid; I had a normal temperature several times during the course of these twenty-one days, although I gave a prompt leukopenic and a prompt Widal reaction, and I had the complications which are typical of typhoid fever, such as a phlebitis of the left external saphenous vein, which almost of itself is enough to establish a diagnosis of typhoid fever in a fever of that type running twenty-one days. Of course, I attributed the partially afebrile course to this vaccination.

I think that the typhoid vaccines can be used with advantage, provided they are used *before the stage of invasion of the blood* by the germs in any amount. I think, however, that it is perfectly useless to attempt to introduce the vaccine after that stage, because by that time blood cultures are positive. If results are to be obtained, they must be obtained before a generalized infection of the vascular apparatus has taken place.

Dr. Murphy: You are giving this man an injection, today, in what part of the body?

DR. MIX: I shall give him 500,000,000 in the left arm. It does not matter, of course, where the injection is made—whether in the abdomen, the arm, or in the thigh. The arm is a very convenient place, and it will be given there in exactly the same way as one would give an ordinary hypodermic injection of morphin.

DR. MURPHY: Let the records show that Dr. Mix injected 500,000,000 dead typhoid bacilli subcutaneously into the arm.

I want to show you this young man with typhoid spine, and I want to call your attention to some of the things in connection with typhoid spine. This is not purely a case of typhoid spine, and that is a point I wish to accentuate. This boy has had repeated attacks of the "rheumatoid" type of spondylitis preceding this trouble. I say "rheumatoid," because it is supposed to mean more than rheumatism. It is only a nicer way of saying it; it does not mean any more. It means nothing.

The only place in which we could locate his primary infection was in his nose. He has had a continued catarrh. In one of our cases here the antrum was infected, then the frontal sinus; then the other antrum was infected, and then there were "rheumatoid" metastatic infections in many of the large joints. This boy did not have the sinus infections. An acute infection of his nasal mucosa was followed years ago by an infection in his spine. Now he comes here. He has no frontal or antrum infection now.

It is peculiar that from these mucous membrane infections we get these constitutional manifestations in the joints. We occasionally have them follow cutaneous infections, as we noted in the case of the man who had a furunculosis, and in whom a typical furunculous metastatic perinephritic abscess appeared. The infecting microörganism in both cases was the same type of staphylococcus, showing how easily these metastatic infections can be traced to their primary source.

In the course of typhoid, inflammation in the vein does not appear until eight or ten days, as a rule, and often not until twenty-five days, after the onset of the disease. You do not get a typhoid deposit in the bone as a typhoid osteal abscess, nor do

you get typhoid arthritis until in the later stages of the disease, usually after twenty-one, twenty-two, twenty-three, or twenty-five days. Dr. Mix had phlebitis much earlier than usual when he had his attack of typhoid. There was a definite reason for it. Dr. Mix was compelled to remain out of bed on account of the intense pain in his back. He sat up in an easy chair much earlier than he should have done, and consequently he had to pay the price. That was the element in producing phlebitis so early in Dr. Mix's case, because we know that phlebitis occurs late in the other cases, and the other patients are practically always kept in bed, and are not permitted to get out of bed until after a certain length of time has elapsed.

QUESTION (TO PATIENT): How much trouble did you have with the spine or with the joints before you had typhoid?

PATIENT: I had considerable trouble two years ago. I was working pretty hard, and lifting was a good part of my work. I noticed I was stiff. It was hard for me to turn over in bed. I was stiff in the morning, and it was hard to straighten out. After I once straightened out and started on my work I limbered up, and I did not notice it again until I was in bed at night.

DR. MURPHY: When you began to work, how long was it before you noticed the first trouble with the back?

PATIENT: It preceded my work.

DR. MURPHY: The work accentuated the trouble in the back?

PATIENT: I think so.

DR. MURPHY: You do not know whether your spine was fixed at that time or not?

PATIENT: No.

DR. MURPHY: His spine is badly fixed, yet it is questionable whether it is ankylosed or not. The spine may become just as fixed as it is in this patient in the acute process without ossification of the intervertebral cartilages and without ossification of the perivertebral connective tissue. When the disease has advanced, as was shown beautifully in a specimen which I have in my office that came from Vienna, you not only have complete ossification of the intervertebral cartilages, but periosteal ossification, or ossification of the deep fibrous ligaments which connect one ver-

tebra with another. It is typical of spirochetic disease, and is known as a Charcot joint, with the production, as well as destruction, of bone.

This patient has a negative Wassermann, so that we have here two conditions: (1) Involvement of his spine preceding his typhoid; (2) exaggeration or an increase in the spondylitis by an infection from his typhoid, and probably a second deposit of the typhoid bacilli in this entire zone.

In the management of this case we have outlined a definite plan: First, to neutralize the typhoid element by the injection of a large quantity of dead typhoid bacilli; second, after that is stopped, if we have no place where we can get an autogenous vaccine, we will use pyocyaneus vaccine. The pyocyaneus vaccine has the effect of increasing the number of lymphocytes and increasing the number of leukocytes generally. It also has a decided beneficial influence on this class of cases.

The best influence we get, however, is from the injection of autogenous vaccines.

We had a man under our charge who had his primary infection in his bladder. He came in with a typical "rheumatoid" arthritis. He had colon bacilli and Staphylococcus albus in his bladder, associated with a severe cystitis and a continuous discharge of pus, following the removal of the prostate.

At the end of some weeks one joint after another became involved, until practically every joint in his body was invaded by the infection. He was put on autogenous vaccine, first 50,000,000, but that had little effect. We increased the dose to 100,000,000, to 150,000,000, to 200,000,000, and then he commenced to get a reaction and improved.

The patient was sent to us with the idea that there was no hope for him. His case was one of typical "rheumatoid" arthritis, and it was thought that the process would go on until finally all his joints would become involved and anky osed, and he would be completely incapacitated. We believe that, with the treatment he has received, he is going to make a full and complete recovery—that is, the autogenous vaccines are doing the work. He is taking, in addition to the autogenous vaccines, treatment

which aids his elimination; he takes hot baths and electric baths. He is in Hot Springs now. We believe that the greatest benefit is to come from the use of the autogenous vaccines. He has also had from three to five injections of 2 per cent. formalin in glycerin in all his joints.

One cannot escape from the fact that the typhoid vaccine has had a great influence in a prophylactic way. Its specific influence is as great as that of antitoxin in diphtheria. Again, one cannot escape from the fact of the positive prohibitive effect of injections of the antitetanic serum. We have not had a single case of tetanus occur in this hospital since we began using immediate injections of antitetanic serum, running over a very large number of cases of different types of crushing injuries, and the same class of injuries from which we formerly had one or two cases of tetanus every month. The reason internes in this hospital have no experience with tetanus is that they do not know what tetanus means from observing its evolution in cases treated, except when a patient is brought in who had tetanus before the antitetanic serum had been used.

We give the antitetanic serum at the time of the injury. It has little, if any, effect once the disease has developed, and it is not intended to cure. It is intended as a prophylactic, and not as a curative method of treatment.

A few fatal cases of anaphylaxis are recorded in the history of antitetanic serum injections. Asthmatic patients should be questioned closely concerning the etiology of their "asthmatic" spells.

EXTRADURAL HEMORRHAGE FROM TRAUMA: EXCISION OF THREE AND ONE-HALF INCHES OF DURA

HISTORY

MALE, twenty-five years of age, entered hospital March 11, 1912, because of symptoms resulting from a blow on head thirteen years ago.

Family history negative. Was always very healthy until the age of puberty, when injury occurred.

Present Trouble.—In February, 1900, when thirteen years of age, he was struck on the left side of the head, 4 inches above the ear, by a strong, fifteen-year-old boy, using a piece of board 4 feet long by 6 inches wide. His father thinks that he was rendered unconscious for five or ten minutes. He was carried to his home. He had no headache. A surgeon who saw him sewed up a small scalp wound and pronounced the injury not serious. Next day the child was apparently as well as usual.

He was always a bright, active, talkative boy. He never stammered, nor was he unusually quiet or backward in any way. Following the injury he attended school and carried his work with credit. Seven months after the injury his mother noticed that he was becoming more nervous, more easily excited, and more irritable than usual.

During 1902, two years after the first injury, he fell backward from a cart on which he was riding, falling a distance of 4 feet, and striking on the back of his head on the unpaved roadway. He was rendered unconscious for about two minutes, but was able to climb back into the cart and ride home. The scalp was not wounded, and no ill results were apparent. This second fall seemed to affect his nervous condition and change his disposition.

He became irritable—very much so at times. He was easily

excited and talkative. His mother noticed that while talking rapidly and easily, as he always did, he would occasionally halt in the middle of a sentence as if for a lost word. This continued and very gradually became worse, and, unlike his natural self, he became embarrassed easily. His brother discovered, while at school, that he became slower and more labored in his answers. Finally he could not keep up in his work and quit school altogether.

During 1904 his right hand and wrist became weakened, and he could not use them as usual, though at no time did the hand become completely paralyzed, nor were there any movements which he could not make if he continued the effort long enough. No atrophy has ever been apparent.

Four years ago he began to have trouble in swallowing, and while eating would choke frequently. The face seemed more flaccid on the left side, and when he smiled the angle of the mouth would pull to the right somewhat. Saliva often would drool from the angles of the mouth.

At about the same time that difficulty in the face began his father thinks his right toe and leg dragged slightly as he walked. At no time did this become pronounced.

He has complained of a giddy, dizzy sensation in his head often, but he never has had a headache, vomited, or become unconscious, nor has he ever had convulsions or muscular contractions of any type. During the past two years he seems more irritable, and at times is unmanageable. At these times he becomes angry, and then he moves and talks better than at any other time.

His doctor states that eye-ground examination has repeatedly proved negative. Eye reflexes are normal. Sensation is perfect over the entire body.

Laboratory Findings.—Urinary examination proved negative.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix will give you some points in connection with the history of the case of this boy.

Dr. Charles L. Mix: An examination of this patient gave

approximately the following findings, from a neurologic point of view: As to the reflexes: His tendinous reflexes, his bicipital and tricipital, and those of the extensors of the forearm were all exaggerated; the patellar and the Achilles jerks were also exaggerated. The pupillary reflexes, both as to light and accommodation, were normal. The epigastric and abdominal cutaneous reflexes were absent; the plantar reflexes were present and normal; there was no abnormal reflex on the part of the upper motor neurons, that is to say, there was no Babinski, Gordon, Oppenheim, or Chadwick reflex to be obtained. As to sensory disturbances: I put a sharp-pointed pin through the skin of his back without much manifestation of pain on his part; but on account of a defective mentality, we are unable to say whether there is any real disturbance of sensation or whether there is rather a disturbance of apperception. He had, on the tip of the left forefinger and of the left third finger, burns from matches, which might possibly suggest syringomyelia, but when one examines him further he is certain that these burns were accidental by reason of the lack of appreciation rather than by reason of any interruption of any of the conductivity of nerve impulses. He also has a scar from a wound on his left forearm, this lesion having been sewed up by Dr. Brooks without much manifestation of pain by the patient. Owing to his mentality and to his inability to speak we could not, by the simplest language, induce him to indicate to us anything about his muscle and joint sense, his tactile, temperature, and pain senses, so that this question of sensory disturbances is left, so to speak, unsettled.

As to atrophies, a very important neurologic sign: In looking over his scapular muscles, the suprascapulars and infrascapulars and the rhomboidei, we found no atrophies whatever. Both the right and left trapezius muscles were well nourished. His humeral group, triceps and biceps on both sides, are of equal development. As to the forearm group, both the extensors and flexors are developed alike on two sides, and there is no atrophy of the muscles of the lower extremities or the trunk, the back muscles being fairly normal except for a mild degree of scoliosis.

As to motor disturbance: He has spasticity of the right arm

and of the right forearm and right hand, and he also has spasticity of his lips and of his tongue. Besides these *spastic* disturbances, there is a *paretic* disturbance of some of the muscles. He has spastic paresis of his right arm, forearm, and shoulder-girdle group of muscles, and of the right side of the tongue and of the facial muscles about the mouth. He has, however, the power of grasping. His lower extremities show nothing; they are absolutely normal, without spasticity or paresis. He walks perfectly well, without spasticity, paresis, or ataxia. He has apparently no intention tremor in his hands. When you have him make the test-tube test, that is, pour water from one tube into the other, he does it without spilling a drop of water. It is not truly an intention tremor, but a spastic tremor, when carefully analyzed. It is on account of that spasticity that he cannot button his clothes and cannot dress himself.

He is depressed, he is emotional, and he cries easily. His finer sensibilities have gone by the board to a slight extent; he wets his trousers without being particularly disturbed about it.

There are some negative points. There is no scanning speech, no nystagmus, no Romberg sign present, no ataxia of the lower extremities, and no ataxia of the upper extremities.

As to the conclusion to be reached in this case, the first question is this: Is the spinal cord affected in any way? If it is, we have to entertain the possibility of multiple sclerosis. There is no sign of involvement of the spinal cord from top to bottom. There is no atrophy in any part of the spinal musculature, and no disturbance in the anterior horn cells. There is no abnormality in the reflexes of the cord to indicate that there is anything the matter with his spinal cord. There is no loss of the bladder control—in short, nothing to which one can attribute his malady so far as the spinal cord is concerned.

The next question is, Can you charge up any of these things to his medulla? Is the disturbance a medullary one, or is it one that is far forward? When one finds a medullary lesion affecting the lips and tongue, suggesting, as does this case, the diagnosis of bulbar palsy, one ought to find, associated with deviation of the tongue, atrophy of the same side. But he has no atrophy of that

side of the tongue; therefore there is no real reason for believing the disturbance of paralysis with deviation of the tongue and of the paresis of the lips, without atrophy, to be due to bulbar disease. Moreover, if the disease were really bulbar paralysis, with the twelfth and seventh nerves involved, why should the intervening nuclei escape ? Moreover, there is no vertigo. Most of these medullary cases show vertigo on account of involvement of fibers running up into the cerebellum through the cerebellar peduncles. He has none of these. The conclusion is that there is no medullary trouble.

There remains for consideration the cortex. With the history of injury which we have in this case, the most sensible diagnosis is that of subcortical cyst, which would easily follow such a trauma, and which would be responsible for the set of findings we have, or it may be due to a thickening of the dura over the motor area supplying the right arm and the speech-center. Since many of these cases of early trauma are later followed by cysts, the finding of a cyst would not surprise us when the operation is done.

Now, of course, in arriving at a conclusion, I did not have the benefit of an *x*-ray plate. An *x*-ray has been made since I examined the patient. The differential diagnosis, when I saw the patient yesterday, was between multiple sclerosis, bulbar paralysis, and organic disease of the brain cortex, and we decided in favor of the latter. With the *x*-ray plate I think there is additional reason for the conclusion that the motor cortex is diseased or compressed by dura thickened from trauma.

The next thing to do is to resort to trephining and to the clearing up of the situation, so far as it can be done; but, of course, if there be disturbance of the underlying cortex of any grave amount which has lasted over a course of twelve years, we cannot expect to have restoration of that cortex to the normal.

Dr. Murphy: What Dr. Mix has said gives us a basis for action, and the line of investigation we have carried out in this case should be carried out in all cases. Dr. Mix has had an exceptional experience with lesions of the nervous system, consequently his opinion is worth more than the surgeon's opinion in the diagnosis of these lesions, whatever they may be, and he has

outlined the various symptoms to you in a very lucid manner. On that basis, and with the *x*-ray findings, there is only one conclusion to draw in this connection, and that is there is a possibility, if not a probability, of our being able to do something for this patient in a surgical way. This boy is becoming more irritable, so that it is very trying to his parents to put up with him. We do not know whether he has a subdural cyst or not until we have operated. We know, in the epileptic cases, after removing a portion of the indurated and adherent dura, it lessens the irritability of these patients often for months and even years, and that has been our plan of management in this particular class of cases.

You remember the case we operated on a week ago today. That man is now able to move his arm better than at any time. He is able to throw the arm around. His speech has improved materially, and yet it is only a week since we took out a cicatricial mass. He is better now than at any time since he received his injury, which shows the significance of removing the constant source of irritation.

The other type of lesion which Dr. Mix alluded to, thickening of the dura due to a primary pachymeningitis or to a subcortical cyst, comes on after injuries, grows slowly, finally attains considerable size, and drives so many of these patients into an asylum. It is one of the things that has been overlooked by surgeons and physicians until very recent times. You doubtless remember the splendid paper written by Dr. Dudley P. Allen, of Cleveland, two or three years ago, in which he analyzed 12 or 15 of these cases, following comparatively mild traumatisms, most of them not sufficient to render the patient unconscious, this condition developing under the scar. This boy, I understand, has never had convulsions. Here we have a case for operation on the recommendation of the medical man as a probability—better, as a possibility—of doing something for relief. In this case there is absolutely nothing to lose. Death is preferable to his present condition. The boy is becoming very vicious in his thoughts and in his actions, and so pronounced are these things that he has to be separated from his family. At first there was

Fig. 82.—Skiagram showing apparent thickening of skull on the right side.

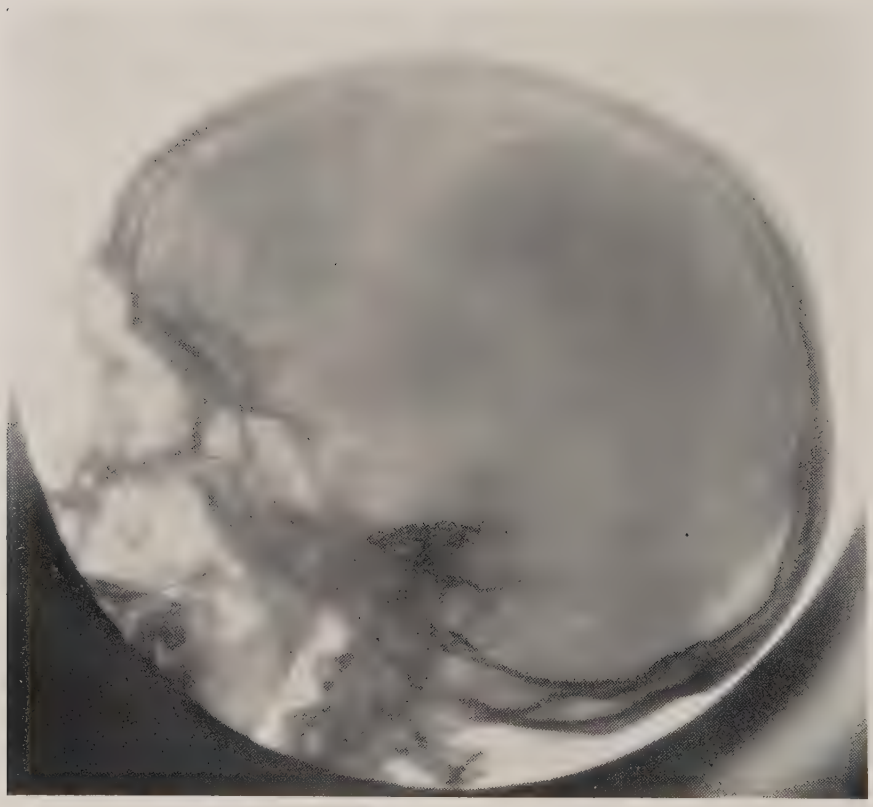

Fig. 83.—Skiagram showing shadow posterior to sagittal suture just below vertex.

none of that. Those things occasionally occur, but not often, in the epileptic, due to local irritations from a fracture.

We will make the usual exposure by making a U-shaped incision for the purpose of raising a skin, bone, and dural flap, 4½ inches broad and 3 inches long, from the occipitoparietal zone, so as to expose the Rolandic area. The hemorrhage is controlled by a rubber tube constrictor. The skull is opened with an electric trephine of Dr. J. M. Neff, and the electric craniotome, also devised by Dr. Neff. The skull is thick—very materially thicker on the anterior margin at the position in which he received the greatest force of the original injury. The bone does not fragment like the other bone, showing that its vascularity has changed. We are making this large horseshoe-shaped flap because any operation which will avail must avail by a large exposure. This deVilbiss instrument, I think, next to the Neff instrument, is the best instrument we have for this work. The advantage which the deVilbiss has over the Neff instrument is that it works more regularly. The instrument is of disadvantage because it requires two or three times the amount of work, and many more units of physical energy, on the part of the operator.

Now I have made this large horseshoe-shaped flap, and will narrow it near the lower end so as to control the line of fracture. We will not go down deep into the fossa, as we can separate the dura quite a distance below the line where we fracture the bone.

We detach the dura over the entire zone to which we wish to have this fracture expose the brain. Now we have a fairly good exposure.

There is no tumor mass to be felt. The brain is very soft right over his speech-center. See how that depression stays where I put in the handle of the scalpel! It is just where he was struck. We take out all that zone of thickened dura to stop that irritation. I cut that dura so that the margin of the dura comes inside the bony portion of the flap all the way around, so you can get so much decompression.

He probably had a subdural hemorrhage which gave him this softening that he has in this position, and it is a lesion of trauma, rather than a lesion from a constitutional condition, which was

also our conclusion, and this accentuates the significance of the early management of brain traumas and what should be done with them.

For a great many years—not originally our own suggestion, but on the suggestion of that master mind, Dr. Fenger, who was so prominent in the last century—we have followed the plan of his original work. In every skull injury where the patient has been momentarily stunned to a degree to render his recollection indistinct, we have put the patient to bed, insisting on the maintenance of the recumbent position for a period of at least six weeks. By doing that we believe that we have secured better results than we did by the former plan of management, namely, allowing them to be around immediately after the effects of the injury had passed.

From our knowledge of the histology and pathology of the brain we find that in concussions and contusions it requires the brain a longer time to get its normal circulation restored than it does any other tissue, and for that reason, whenever the brain is concussed or contused, it demands a long period of rest to have repair take place with the formation of least connective or cicatricial tissue.

If you have a broken leg or a bone injury in any other position of the body where you can see the lesion, you endeavor to keep the patient quiet during the process of repair, so as to have the parts approximated as accurately as possible, and to get restoration of the tissue with the least intervention of newly formed cicatricial connective tissue. Wherever you have a large quantity of newly formed cicatricial tissue you have contraction; wherever you have contraction you have irritation, and this is particularly injurious in the brain. We believe in the plan of Fenger and have followed it for a long time. It requires a great deal of perseverance to control patients, and it requires a great deal of persuasion to show them they are in error if they endeavor to get about before the process of repair has made sufficient headway.

Every interposition of connective tissue is a detriment to the function of the organ or viscus after repair has taken place, so

that it is an important factor in the early management of these brain cases.

What are the dangers that occur in connection with this type of injury, namely, the blow which this boy received on the side of his head, anterior to the middle meningeal artery, and high over the upper portion of the tip of the anterior branch of this artery?

The dangers are these: *First*, the effect of the concussion, an immediate communication of the disturbance to the cell circulation; *second*, a disturbance in the circulation of the brain or meninges, such as laceration of the middle cerebral or meningeal artery.

The average case of rupture of a middle meningeal artery has a typical cycle or course, and I cannot do better than mention here the clinical history in the case of a man who was playing polo. He was struck by the ball on the temporal region. It unhorsed him; he got up dazed, remounted his horse, rode about a bit, but did not enter the game. He went home; on his way he felt dizzy and had a headache. He was put to bed, and little attention was paid to him for an hour or two, when it was discovered that he was completely unconscious, and then, of course, the alarm was given, physicians were called, and a diagnosis made of rupture of the middle meningeal artery. The former plan of management for that was attempted—sending to the city for a doctor, as he was in a suburban town. The doctor came there with all haste, with all his instruments, and in the mean time the patient had gone into a profound stupor. Before this doctor saw him he was seen by a very distinguished doctor, who also believed the trouble would amount to nothing, because he was a little stupid in his responses, complained of his headache, and the doctor said, "I believe it is just a slight concussion and means nothing more." When the second doctor arrived with his instruments he did a trephining operation. By that time a large clot of blood had formed from the ruptured middle meningeal artery, and the patient never rallied from the operation.

I simply cite that to show you the cycle in the course of the symptoms. First, after the immediate shock, they get up and

walk about, as a rule. They are fairly clear in their intellect, and get along nicely, and then gradually become unconscious. That is practically pathognomonic of rupture of the middle meningeal artery, either intradural or extradural.

All you need to do when you feel that the patient is going into a state of coma on account of rupture of the middle meningeal artery is to make a little incision under cocain anesthesia, salt solution, or sterile water analgesia, cut down on his external carotid, and throw a ligature around it. That stops the hemorrhage, and you are master of the situation immediately. The middle meningeal artery is a branch of the internal maxillary, which is a subdivision of the external carotid. It has a terminal circulation, so that one ligature stops the bleeding. You get him out of danger at once, and you have not hazarded the patient at all. It is a simple matter, and uniformly overlooked in the management of these cases. A diagnosis can practically always be made, as they all have some return of consciousness after the concussion. You have a contusion or depression at the site of injury, which tells you exactly what has happened. After ligating the carotid one can take his time to do the trephining.

Let the record show that there were no adhesions to the dura; that there was a large, softened area, as large as a bantam egg, just at the lower end of the fissure of Rolando; that a depression of the pia there left a concavity that it took several seconds to fill. There was no tension on this, and for that reason it was not thick. There was an increased vascularity in the veins over that zone and over all the zone from which the dura was removed. That a piece of dura, 3½ inches long by 2½ inches wide, was entirely excised; that this was not excised clear to the margin of the bony elevation—I left ½ inch of the dura on which the edge of the replaced bony flap could rest; throughout the entire circumference of the exposed zone there were no adhesions to the pia; that there was some inflammatory process produced within the brain substance itself around the speech-center which continued to a softening and degeneration of the brain following trauma; that there was an extradural hemorrhage from trauma which caused a cicatricial formation in the dura.

What we hope to secure here is not the restoration of function in this zone, but a suppression of the irritability, which is often obtained when we excise the dura; if we accomplish that, we will do well.

The patient convalesced without untoward symptoms. There was no material change in his general condition up to the time he left the hospital, five weeks later.

POTT'S FRACTURE

HISTORY

PATIENT, a girl aged fifteen, admitted to Mercy Hospital January 5, 1912. Family and personal history were negative.

Present Trouble.—On June 8, 1911, the patient stepped on a stone; her foot slipped and was forcibly everted. She fell to the ground and could not rise. The ankle and leg were much swollen, red, and intensely painful. The leg was placed in a fracture-box at once, and twenty-four hours after the accident a mold was applied and left on for six weeks. When it was removed the ankle was deformed and the foot was everted. The patient has been able to walk since, but limped. There was visible shortening of the leg. She could not hold the foot in a straight line with the leg.

COMMENTS AND OPERATION

Examination.—The proximal portion of the tibia was overriding internally the base of the internal malleolus, and the latter was directed downward and outward instead of backward and inward. The external malleolus was broken off and displaced outward and also upward on the outer surface of the fibula. A plumb-line drawn through the middle of the tibia passed just to the inner side of the articular surface of the astragalus, showing the outward luxation of this bone. There was a pronounced abduction of the foot. It was a classic superlative deformity from a Pott's fracture.

Operation.—The internal malleolus was exposed by an incision to the inner side of and parallel with the tendon of the tibialis anticus. The line of fracture at the base of the malleolus was clearly demonstrated. It was refractured a little higher up, and on a line directed upward and inward from the articular

surface, as so to permit of sliding upward when the deformity was reduced. The external malleolus was then exposed and divided on a line more slanting upward and outward. The newly formed interosseous ligamentary attachments were freed, and the foot was displaced inward. The internal malleolus slid upward on the inner surface of the tibia, and the external malleolus was slipped downward on the outer surface of the fibula. The internal malleolus was then fixed with one nail driven upward and outward, and the external malleolus was fixed with two nails, so that the midline of the articular surface of the astragalus was held a little to the inner side of the midline of the articular surface of the tibia. The foot was dressed in an adducted position and flexed on the tibia. Primary union resulted. The mold was removed at the end of four weeks, when motion in the ankle was perfect and painless. Seven weeks after the operation the nails were removed. Complete result. Complete bony union with a little thickening of both the external and internal malleoli, due to the callus around the large, freshened bony surfaces made during the operation. The patient stands plumb on her foot, and has perfect motion on leaving the hospital, March 7, 1912.

Fig. 84.—Pott's fracture. Photograph showing deformity before operation.

Fig. 85.—Pott's fracture. Photograph showing deformity before operation.

Fig. 86.—Pott's fracture. Skiagram showing Pott's fracture before operation.

Fig. 87.—Pott's fracture. Skiagram showing Pott's fracture before operation.

Fig. 88.—Pott's fracture. Skiagram taken through the cast two weeks after operation, showing fragments held in position by the nails.

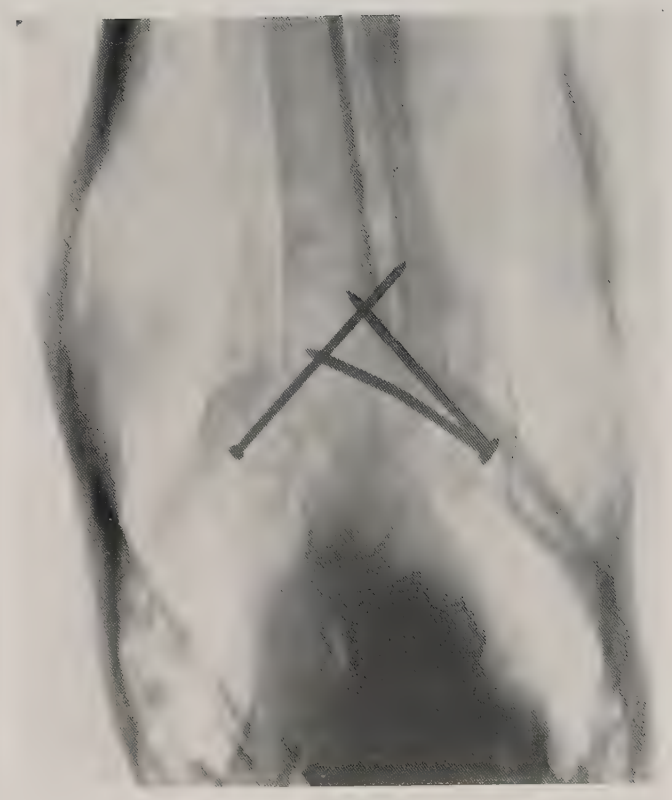

Fig. 89.—Pott's fracture. Skiagram taken through the cast two weeks after operation, showing fragments held in position by the nails.

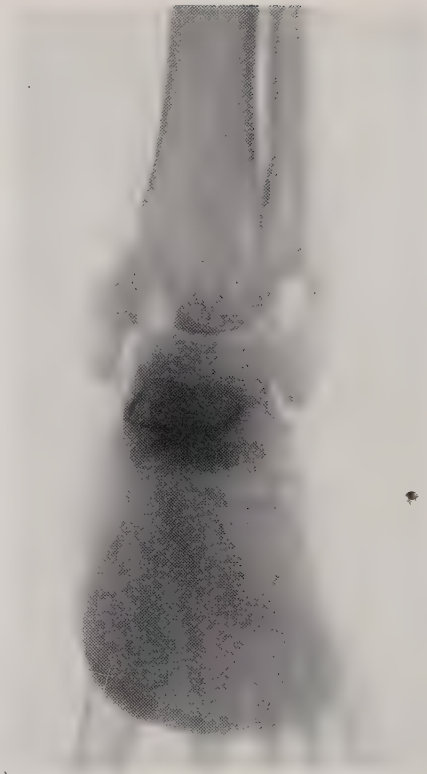

Fig. 90.—Pott's fracture. Skiagram taken after removal of nails two months after operation. Bony union between each malleolus and its respective shaft. Astragalus in plumb-line with articular surface of tibia.

Fig. 91.—Pott's fracture. Skiagram taken after removal of nails, two months after operation.

Fig. 92. — Pott's fracture. Photograph taken when patient left hospital, and showing results of operation. Symmetry restored.

Fig. 93.—Pott's fracture. Photograph taken when patient left hospital ten weeks after operation. Symmetry restored.

FIVE DIAGNOSTIC METHODS OF JOHN B. MURPHY*

In the every-day work of a large surgical clinic, where there is maintained an aggressive spirit of progress, new ideas and new methods are constantly being tried, and deductions made as to their value. Those proving of no merit are soon discarded, but those of worth are adopted, and in time become a part of the regular routine procedure. The longer the accepted methods are used without showing signs of being fallacious, or without being superseded by better ones, the more weight is given them. This is true in the matter of operative technic, but it is still more true of diagnostic methods, and the latter, if possessing real merit, are of value not only to the surgeon, but also to the general practitioner.

In the service of Dr. J. B. Murphy there are in use five diagnostic methods which were original with him, and which have been used by him for a great many years. They have been thoroughly tried out, and have proved their unquestioned value as aids in the differential diagnosis of some of the pathologic conditions within the abdomen. Their reliability and simplicity of execution make them easy of application and increase their usefulness, especially to the physician in general practice.

The first is what has been called by Dr. Murphy the "fist percussion of the kidney," and is used to determine the presence or absence of an acute pathologic condition within that organ. As shown in Fig. 94, the patient, with clothing removed above the waist, is seated in an upright position on a stool, and is then

* [*Editorial Note.*—Dr. Murphy, in his clinical talks, constantly refers to the "five diagnostic methods" described in this article. In order that our readers may thoroughly comprehend these we print the article complete as it was published in the "Archives of Diagnosis" in January, 1910. The article was written by Dr. Guy G. Dowdall, at that time a member of Dr. Murphy's staff.]

instructed to bend forward as far as possible. With the patient in this position the examiner, from behind, places his left hand flat upon the back of the patient over the kidney of the one side or the other, care being taken to have the hand pressed firmly upon the back. The clenched right hand of the examiner is then brought down with considerable force upon the dorsum of the fixed hand (Fig. 95), and if an acute congestion, infarction, retention in the pelvis of the kidney, or ureteral obstruction exists in that kidney, the patient will cry out with the pain of the blow. It is analogous to striking a distended urinary bladder, and if the pathologic condition be present, the result will be decidedly apparent. A good sharp blow is used, but it is not necessary to use force enough to aggravate any diseased condition within the kidney. A control test is advised by first using the blow on the sound side and then on the suspected side.

A second method, and one depending upon the same principle as the preceding one, is used by Dr. Murphy in his examination for an acute infection of the gall-bladder, or acute obstruction of the cystic or common duct, with or without infection. The examiner, sitting at the right side of the recumbent patient, presses the tip of the second finger of the left hand, flexed at a right angle, firmly up under the costal arch at the tip of the ninth cartilage, as shown in Fig. 96. The patient is instructed to take a deep breath, and at the height of the inspiration, when the gall-bladder is forced below the costal guard, the flexed finger is struck forcibly with the ulnar side of the open right hand of the examiner, and if there be an inflammation or a retention in the biliary tract, the patient will announce that the blow caused him severe pain. One is again striking an overdistended or inflamed viscus that contains extremely sensitive nerve-filaments. Dr. Murphy calls this the "hammer-stroke percussion." The force used in striking the finger is well shown in the illustration (Fig. 97), for here one sees that sufficient force was used to sink the finger well up under the costal arch.

Another method, designated by him as the "deep-grip palpation," is somewhat similar to the above, and is also used in an examination of a suspected gall-bladder disease. It is based

Fig. 94.—"Fist percussion of the kidney."

Fig. 95.—"Fist percussion of the kidney."

Fig. 96.—"Hammer-stroke percussion."

Fig. 97.—"Hammer-stroke percussion."

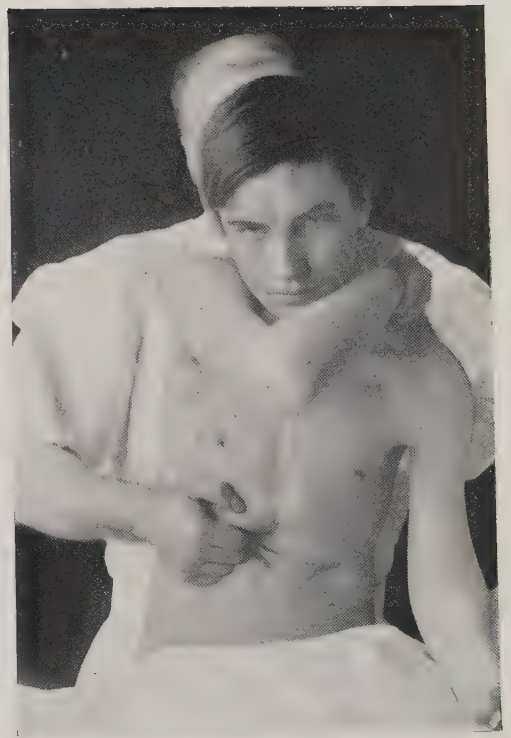

Fig. 98.—"Deep-grip palpation."

Fig. 99.—"Piano percussion."

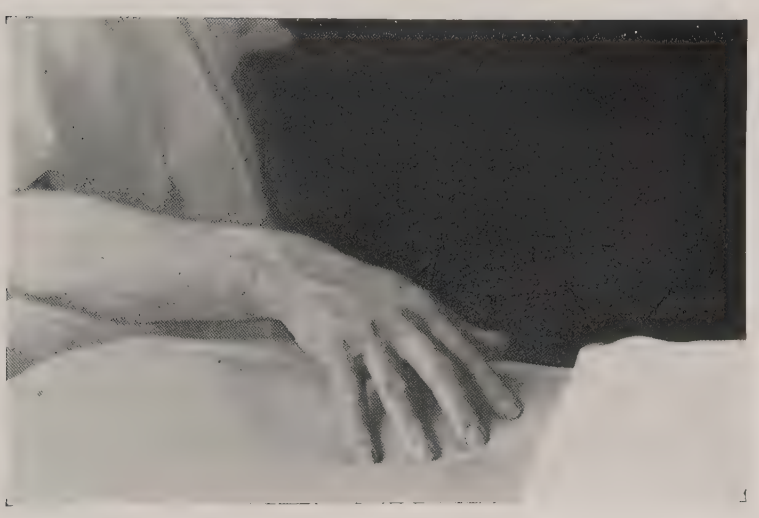

Fig. 100.—"Piano percussion."

upon another mechanical method of squeezing the gall-bladder, depending upon the diaphragm to tell the story. Standing directly behind the patient, as in the accompanying photograph (Fig. 98), provided the patient is well enough to assume an upright position,—if not, the examiner reaches over the recumbent patient from the head,—the right hand of the examiner curls up under the costal arch at the tip of the ninth cartilage, the patient is requested to take a deep breath, and, at the end of the expiration, the examining fingers seek the gall-bladder area and fix it from beneath. The diaphragm, descending from above with the beginning inspiration, brings down the liver and gall-bladder, and if a cholecystitis or cholelithic obstruction is present, the descent is checked suddenly with an accompanying groan from the patient, for the diaphragm, acting as the great piston of respiration, forces the distended, inflamed, or congested gall-bladder down upon the firmly fixed hand, and the result is an abrupt cessation of the inspiratory act, plainly shown by the patient, for he feels a severe pain.

Still another method used by Dr. Murphy is called by him the "piano percussion." It is used to demonstrate a small quantity of fluid or exudate in the abdominal cavity, resting on the hollow viscera, the intestine, and is based upon the peculiarly characteristic percussion-note given off when the abdominal wall over the fluid is struck by the four fingers, one after the other, beginning with the fourth and rapidly going to the index-finger (1, 2, 3, 4, as shown in Figs. 99 and 100). There is a complete absence of the tympanitic note which one gets in this condition when the ordinary percussion methods are used, for with the usual percussion stroke the tympanitic note from the distended bowel so obscures the faint flat note coming from the fluid that the latter is entirely lost. This "piano percussion" is so superficial a stroke, or rather series of strokes, falling almost simultaneously, that only the flat note from the fluid is brought out. It is a somewhat delicate stroke, and requires practice to make the fingers fall properly and in order, but it is of considerable value, and will help to prevent an error in diagnosis.

And, finally, to one other method attention is called. It is

most simple, and should be used by every careful examiner: The simultaneous palpation of both iliac fossæ in cases of suspected acute appendiceal involvement. We have all noted in cases where there is considerable trouble in the appendix that the affected side offers greater resistance to the examining hand. We palpate, first, the healthy side, and then the other, and see quite a difference, especially in typical cases; but Dr. Murphy has pointed out, and proved by clinical results during the past twenty years (since 1889), that decidedly more information is gained when both sides are palpated simultaneously. Compare the way in which the examiner's left hand sinks into the patient's iliac fossa to the lessened descent of his right hand on the side of the inflamed appendix. The difference is grossly apparent in the typical cases. So it is with the lightly involved appendices—those cases with slight recurring attacks—the atypical cases—that this test is most serviceable. An equal pressure on the two sides at the same time, "comparative bimanual examination," will show a difference which, with practice, is discernible in practically all cases where there exists an acute pathologic condition within or around the appendix.

In reporting these methods it is hoped that a working knowledge and use of these procedures will prove of value in that class of difficult cases to which they are applicable, and that it may stimulate also an interest in that most essential field of work—differential diagnosis.

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